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Archives OF OTOLARYNGOLOGY

INDEX NUMBER
IN TWO SECTIONS — SECTION I

THE RATIONAL APPROACH TO IMMUNIZATION OF THE UPPER RESPIRATORY TRACT — THEODORE E. WALSH, M.D.

THE MEDICAL TREATMENT OF MENIERE'S DISEASE — HENRY L. WILLIAMS, M.D.

NASOPHARYNGEAL LYMPHOID TISSUE AND ITS RELATIONSHIP TO HEARING — R. E. JORDAN, M.D.

HYPOTHYROIDISM AS A CAUSE OF HEADACHE — A. CURTIS JONES JR., M.A., M.D.

RADICAL SURGERY ON OS TEMPORALE — TOSHIKAZU NISHIHATA, M.D.

THROMBOCYTOPENIC PURPURA DUE TO QUINIDINE — N. EDWARD NACHLAS, M.D.

VOCAL REHABILITATION OF PARALYTIC DYSPHONIA — GODFREY E. ARNOLD, M.D.

ACUTE POLIOMYELITIS OF THE MOTHER, WITH AURAL LESIONS IN THE PREMATURE INFANT — GEORGE KELEMEN, M.D.

FURTHER PSYCHOSOMATIC ASPECTS OF OTOLARYNGOLOGY — C. STEWART HASH, M.D.

PROGRESS IN ESOPHAGOLOGY — DANIEL C. BAKER JR., M.D., ET AL.

HEARING AND DEAFNESS IN CLEFT-PALATE PATIENTS — EDGAR M. HOLMES, M.D., ET AL.

LOCUS OF THE LESION IN RECRUITING EARS — PHILLIP A. YANTIS, Ph.D.

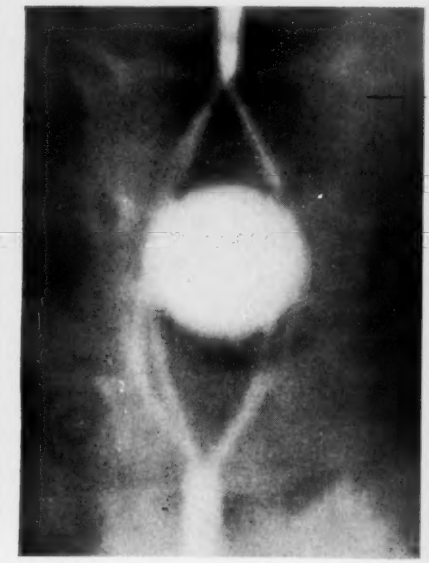
SPEECH AUDIOMETRY BY BONE CONDUCTION — C. P. GOETTINGER, Ph.D., ET AL.

COMPLETE CONTENTS ON FIRST PAGE

DECEMBER 1955

VOLUME 62 NUMBER 6

From Ausband et al., p. 641



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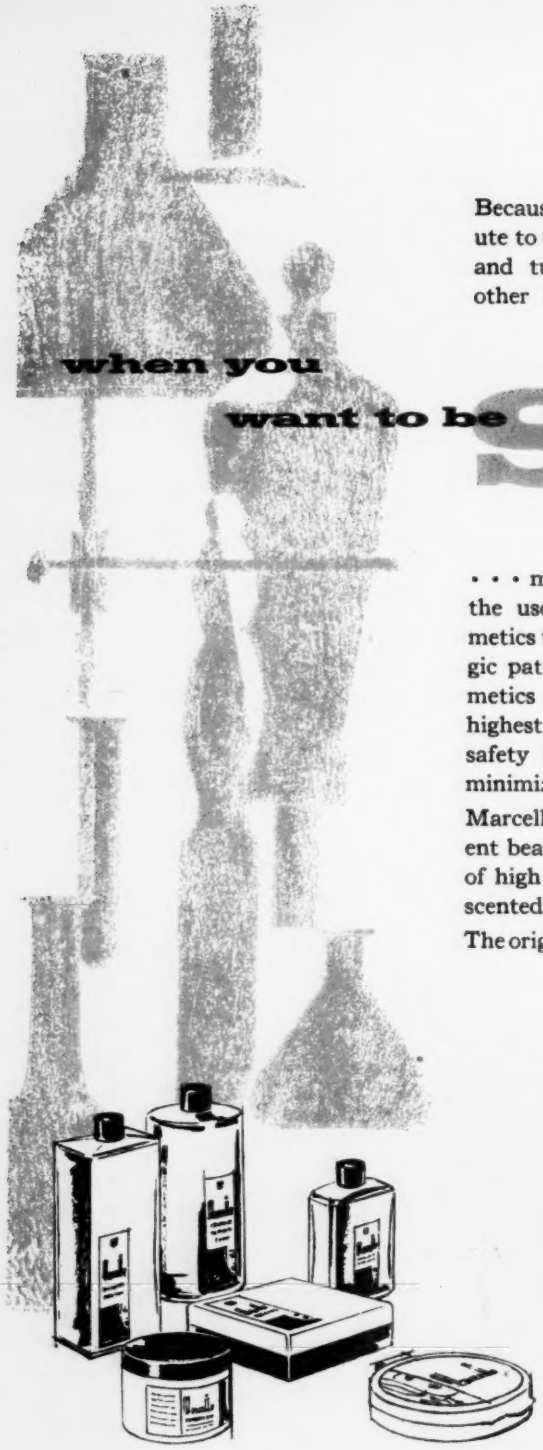

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TABLE OF CONTENTS

VOLUME 62 DECEMBER 1955 NUMBER 6

ORIGINAL ARTICLES

PAGE

The Rational Approach to Immunization of the Upper Respiratory Tract 569
Theodore E. Walsh, M.D., St. Louis

The Medical Treatment of Ménière's Disease 573
Henry L. Williams, M.D., Rochester, Minn.

Nasopharyngeal Lymphoid Tissue and Its Relationship to Hearing 579
R. E. Jordan, M.D., Pittsburgh

Hypothyroidism as a Cause of Headache 583
A. Curtis Jones Jr., M.A., M.D., Boise, Idaho

Radical Surgery on Os Temporale 586
Toshikazu Nishihata, M.D., Tokyo

Thrombocytopenic Purpura Due to Quinidine 591
N. Edward Nachlas, M.D., Baltimore

Vocal Rehabilitation of Paralytic Dysphonia 593
Godfrey E. Arnold, M.D., New York

Acute Poliomyelitis of the Mother, with Aural Lesions in the Premature Infant 602
George Kelemen, M.D., Boston

Further Psychosomatic Aspects of Otolaryngology 611
C. Stewart Nash, M.D., Rochester, N. Y.

Progress in Esophagology 614
Daniel C. Baker Jr., M.D., and Charles A. Flood, M.D., New York

AUDIOLOGY SECTION

Hearing and Deafness in Cleft-Palate Patients 620
Edgar M. Holmes, M.D., and George F. Reed, M.D., Boston

Locus of the Lesion in Recruiting Ears 625
Phillip A. Yantis, Ph.D., Ann Arbor, Mich.

Speech Audiometry by Bone Conduction 632
C. P. Goetzinger, Ph.D., and G. O. Proud, M.D., Kansas City, Kan.

CASE REPORTS

The Surgical Approach to Brain Abscess of Otitic Origin 636
Joseph J. Littell, M.D., Santa Rosa, Calif.

Malignant Schwannoma of Tip of Nose 638
John J. Conley, M.D., New York

An Unusual Foreign Body, a Marble, in the Esophagus 641
John R. Ausband, M.D.; James A. Harrill, M.D., and Hooper D. Johnson, M.D., Winston-Salem, N. C.

Metastatic Hypernephroma Occurring in Nasal Septum 644
M. V. R. Achar, M.B., B.S. (India), New York

SPECIAL REPORTS

Hearing Rehabilitation Centers in the United States 649
Marion P. Downs, M.A., Denver and Eva Carson, A.B., Glenview, Ill.

CLINICAL NOTES, NEW INSTRUMENTS AND TECHNIQUES

A Gastrostomy Tube 665
J. A. Perrone, M.D., Pittsburgh

SOCIETY TRANSACTIONS

Chicago Laryngological and Otological Society 666

REGULAR DEPARTMENTS

Abstracts from Current Literature 675

News and Comment 601, 619, 640, 648

Directory of Otolaryngological Societies 681

A. M. A.
ARCHIVES OF OTOLARYNGOLOGY

VOLUME 62 DECEMBER 1955 NUMBER 6

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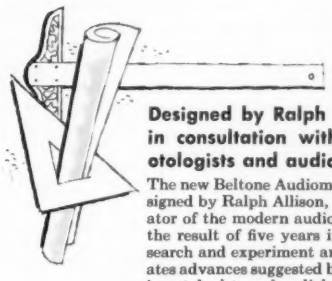

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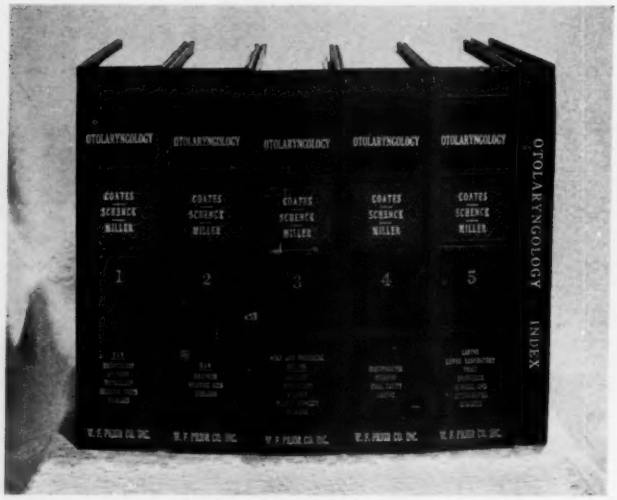

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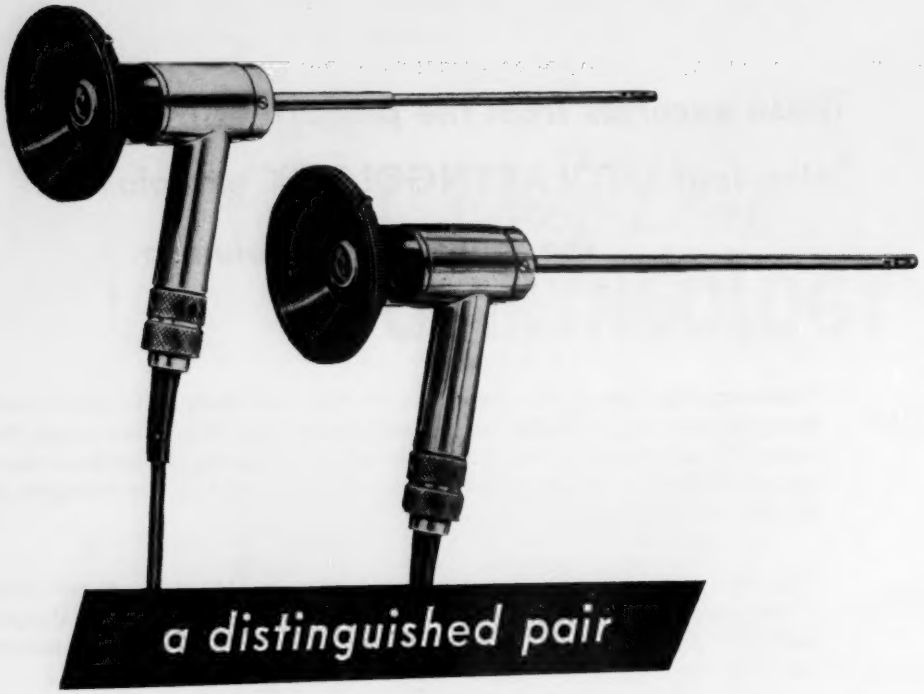

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The Rational Approach to Immunization of the Upper Respiratory Tract

THEODORE E. WALSH, M.D., St. Louis

That a virus is the etiologic agent in many of the infections of the upper respiratory tract has long been recognized. Kruse [1] in 1914, Foster [2] in 1917, and Dochez [3] and his co-workers in 1930 demonstrated that a common cold could be induced in a human subject by the intranasal instillation of filtered washings from the nasopharynx of a subject with an acute cold.

Much work has been done in recent years in an effort further to elucidate the role of the virus in upper respiratory infections. In a recent review article Roden [4] summarizes this work, and the results seem to indicate the following:

1. There are probably several of the viruses responsible for upper respiratory diseases.

2. These viruses are not pathogenic for animals other than man and the chimpanzee.

3. Infections can be transmitted in an experimental human subject in approximately 30% to 50% of attempts.

4. The viral disease when uncomplicated by secondary bacterial invasion is self-limiting and lasts from two days to a week.

Recorded for publication Sept. 13, 1955.

From the Department of Otolaryngology, Washington University School of Medicine.

Read before the Section on Laryngology, Otology and Rhinology at the 104th Annual Meeting of the American Medical Association, Atlantic City, June 9, 1955.

The upper respiratory infections seen by the otolaryngologist are due to the invasion of the tissues of the nose and throat and accessory sinuses by bacteria. The stage for this invasion may be set by a virus infection in some instances or by the breakdown of normal nasal defenses by any of the other factors emphasized by Proetz, by me, and by others. The morbidity due to bacterial infection, however, is far greater than that due to any virus infection. Horsfall [5] states of the viral infections, "If complications do not develop, symptoms seldom persist longer than a week and may largely disappear in three or four days" and, further, "No effective control measures have been devised. Vaccines have not been shown to decrease susceptibility to the disease."

While the antibiotics have been found useful in combating bacterial infections generally, their indiscriminate use has led to the development of resistant strains of organisms, particularly those responsible for respiratory diseases. Davison [6] has emphasized this point and finds, for example, that the doses of penicillin required now to combat infections in the sinuses are many times greater than were once effective. It seems particularly important, therefore, to consider the measures necessary to increase the resistance of the upper respiratory tissues to the common respiratory pathogenic bacteria.

Vaccines, administered parenterally, have been advocated for the prevention of upper respiratory infections. Although benefit has been reported in many instances by many

authors,[7] the results reported by those who have undertaken controlled clinical studies have been disappointing.*

Cannon and I [8] suggested that it was perhaps the method of vaccination rather than indifferent antibody response to the common respiratory bacteria that accounted for these disappointing results. In a series of experiments we † demonstrated the following facts after the local application of a formalized vaccine to the nasal mucosa of rabbits:

1. There was a concentration of specific antibody in the mucosa on an average two to three times greater than that obtained following parenteral vaccination. The ratio of tissue antibodies to serum antibodies (T/S ratio) is on an average 1:10 or 1:15 following parenteral vaccination; whereas, after the local application of vaccine, the T/S ratio is on an average 1:5 and may in certain instances be as high as 2:1.

2. The nasal tissues of animals treated by the intranasal application of vaccine demonstrated histologically an accumulation of monocytic cells (lymphocytes and macrophages) in the subepithelial tissues. These cells were absent in the mucosae of animals treated by parenteral vaccination.

3. When rabbits were treated intranasally with a suspension of killed pneumococci which were highly virulent for the species, they withstood infection by the same organisms by a dose invariably fatal for untreated animals. It was interesting that in animals thus protected against nasal infection the presence of protective antibodies in the circulating blood could not be demonstrated. These experiments suggest that following the local application of vaccine to the nasal mucosa ideal conditions for tissue immunity, i. e., a concentration of specific antibodies and an accumulation of phagocytic cells, obtain.

In 1940 I [12] reported the results of local vaccination of the nose as a preventive measure against upper respiratory infections in susceptible patients. Observations made on 627 patients in an eight-year period showed protection in 84% and no result in 16%. It is interesting that such protection against respiratory infections can be obtained when dead organisms are applied to the nasal tissues, while little, if any, immunity results from an acute infection with the same organisms. As the cellular response to invasion of tissues by living organisms is primarily by polymorphonuclear leucocytes and much later by macrophages, Smith and I [13] investigated the effect of ingestion and digestion by polymorphonuclear leucocytes on the antigenicity of bacteria. We showed that antigenicity was definitely decreased by digestion of bacteria by polymorphonuclear leucocytes.

COMMENT

Although there is evidence that many infections of the upper respiratory tract are due to viruses, viral infections per se are self-limiting and of only minor morbidity. The more serious infections encountered by the otolaryngologist are due to the invasion of the respiratory tissues by pathogenic bacteria. This may occur as a complication of viral disease, but it happens as frequently when the normal nasal defenses break down for other reasons.

In light of the increasing resistance to the antibiotics of many strains of bacteria causing upper respiratory infections, the problem of immunization of the respiratory tract gains importance. It seems not unlikely that unless infection of the respiratory tract can be prevented the necessity for surgical intervention in the treatment of complications will increase. This is already becoming evident in cases of otitis media and mastoiditis.

The experimental evidence points to local vaccination as the rational approach to protection of the respiratory tissues. The method —the daily spraying of vaccines from an atomizer into the nose—requires the cooperation and anamnesis of the patient, as does any other medication prescribed by a physician to be taken at home. It is probable that the immunity conferred by this method is only of short duration—probably from six to eight weeks—and it is therefore important

* References 17 to 21.

† References 8 to 11.

that the application of vaccine be continued during the season of respiratory infections.

The Vaccine.—Because the antibody produced is specific for the organisms used in the vaccine, a polyvalent vaccine containing as many as possible of common respiratory pathogens should be used. It is also important that the vaccine contain sufficient numbers of killed organisms. The recommended turbidity of the final mixture is approximately equivalent to that of 1,000,000,000 staphylococci per cubic centimeter as measured on a McFarland nephelometer scale.

Cannon and I [8] stated that:

> Although the vaccine must pass the epithelial barrier of the nose and pharynx, our observations do not show how this happens. The evidence indicates that particles of vaccine in some way pass the epithelial barriers with the resulting formation or accumulation of specific antibodies in the nasal tissues.

The passage of particulate matter through the epithelial barriers must be a slow process, and it seemed possible that a solution such as Respiratory "UBA" (Lilly) might be more readily absorbed. UBA Lilly is a respiratory vaccine prepared according to the method of Krueger [14] which was later reported upon by Powell and Jamieson.[15] It is said that this method produces an undenatured bacterial antigen which should be efficient in producing antibodies. With the cooperation of Eli Lilly & Company, I ‡ tested the effect of undenatured bacterial antigens prepared from Bacillus typhosus and B. paratyphosus and from the Type I Pneumococcus A-5 and compared the antibody production of these materials against those of the usual vaccines. I found that in no instance could antibodies be demonstrated either in the tissues or in the circulating blood of animals which had been treated with UBA typhosus and paratyphosus in spite of the fact that after the use of the vaccine the usual concentration of antibodies was found. Further, animals treated with UBA Pneumococcus Type I succumbed to a lethal dose of the living organism in every instance, whereas animals treated with the A-5 vaccine were completely protected. It seems evident from these experiments that UBA is not efficient as a vaccine to be used in protection of the upper respiratory tissues.

‡ Walsh, T. E.: Unpublished data.

It is not within the scope of this paper to discuss by what cells antibodies are produced. In a recent article Schenck [16] reviews the literature and makes an excellent case for the plasma cell as being the cell responsible for antibody production. It is of interest that in our sections of rabbit mucosae Cannon and I [8] noted accumulation of macrophages, but we made no mention of the presence of plasma cells after treatment of the animals with local vaccine.

In light of the work of Smith and me it might seem possible that the local application of specific vaccines in the sinuses offers a method of treatment for chronic sinusitis. It seems possible that in a chronic sinusitis after the pus has been evacuated from the sinuses if vaccine were allowed to remain in its place the macrophages in the tissues might be induced to ingest and digest bacterial particles and elaborate specific antibodies to overcome the infection.

SUMMARY AND CONCLUSIONS

1. The morbidity from infection of the upper respiratory tract by bacteria is greater than that from viral infections.

2. In view of the increasing resistance of bacteria to antibiotics, adequate immunization of the nose and throat to bacteria is important.

3. Experimental and clinical evidence points to the use of vaccine locally in the nose as the rational method of acquiring immunity.

4. The vaccine should be a suspension of killed organisms of adequate density.

5. Respiratory "UBA" has proved of no value as a substitute for a suspension of killed organisms.

6. It is suggested that vaccine might prove useful in the treatment of chronic sinusitis.

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The Medical Treatment of Ménière's Disease

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In 1861 Prosper Ménière [1] described a hitherto unrecognized symptom complex characterized by tinnitus, loss of hearing, and vertigo, the latter associated with nausea and sometimes with vomiting and followed by a syncopal attack with cold clammy sweat and weakness. His purpose was to separate this group of patients from the mass of dizzy or vertiginous patients who at that time were lumped together as having "apoplectiform cerebral convulsion."

Unfortunately, Ménière died the year after he described this symptom complex, and his medical descendants soon lost sight of his purpose and reevoked the old confusion by combining all types of vertigo, dizziness, and incoordination under the term "Ménière's symptom complex." However, as Crowe [2] pointed out, the symptoms of Ménière's disease are so invariable that they must originate from a single definite cause and the disturbance, therefore, warrants the use of the term "disease."

The important role that Ménière ascribed to the cochlear signs of the disorder was also lost sight of, and attention was focused on the more spectacular vestibular symptoms and signs. Ménière located the disturbance producing the symptoms he described in the end-organ of the eighth cranial nerve. He based this hypothesis on the physiologic experiments of Flourens.

Further than this Ménière was unable to go owing to lack of facilities for the microscopic examination of the labyrinth, and lack of material for such examination, because the disorder in itself is not fatal. In his résumé Ménière stated that, while the disturbances of function which he described are capable of producing symptoms that could be attributed to disease of the brain, everything led to the assumption that the lesion which is the cause of these troubles lies in the semicircular canals.

As a result of incorrect translation of his papers Ménière was also accused of ascribing hemorrhage into the labyrinth as the pathologic basis of the symptom complex he described, while, as a matter of fact, he was so far from doing this that he was inclined to relate the disorder to migraine.

Great renewal of interest in Ménière's disease was ushered in by the description of the pathology of Ménière's disease by Hallpike and Cairns [3] in 1938. They were fortunate enough to secure material for study due to neurosurgical intervention. Their findings were rapidly confirmed by many other investigators, and attempts to treat the disorder were renewed immediately. Successful medical treatment of a condition is usually based on a knowledge of the etiology of the condition, so that an attempt can be made to return the disturbed physiologic function toward normal. It has been stated that the etiology of Ménière's disease is unknown and that the basis of treatment is purely hypothetic. This statement is partly true, but it ignores a considerable amount of evidence, both direct and indirect, that points to a definite cause.

In the first place, Parrisius [4] and Müller [5] showed that persons with Ménière's disease belong to a group of patients with an inherited constitutional deficiency, a group displaying such disorders as Raynaud's disease, physical and immunologic allergy, acrocyanosis, and the like. They did this by demonstrating by biomicroscopy that when such persons were subjected to a preferred

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stimulus or stress the peripheral vascular bed reacted abnormally: The arterioles of the vascular loop went into spasm, with secondary dilatation and varicosity of the subjoined capillary loops. This reaction is similar to that produced in normal persons by exposure to cold, but in this group it occurs at higher temperatures than in normal persons and can be precipitated by stimuli which are not ordinarily effective in a normal person.

Until recently it has not been possible to view directly the behavior of the peripheral vascular bed of the labyrinth. However, outstanding experimental work by Weille and co-workers [6] has demonstrated the same physiologic mechanism in the labyrinth of guinea pigs subjected to anaphylactic shock as in the nail beds of persons with such conditions as Raynaud's disease. This "spastic-atonic" state of the arterioles and capillaries, as it was termed by Müller,[5] has some stereotyped consequences.

The hypothesis is presented that as a result of the extreme slowing of the blood stream anoxia occurs in the affected tissues. This produces cellular injury, particularly to the vascular endothelium and the blood cells trapped in the dilated capillary loops. This in turn is followed by clumping and sticking of the leucocytes and erythrocytes to one another and to the vessel walls, the so-called blood sludge. As another result of the anoxia and cellular injury, the capillary walls become more permeable and toxic metabolites, such as histamine, are released, tending to increase the permeability of the capillary wall. The result of this is a collection of extracellular fluid of increased protein content and, thus, of greater osmotic pressure. When this train of events occurs in the arterioles and capillaries of the striae vasculares of the labyrinth, a state of endolymphatic hypertension is induced. The endolymphatic hypertension in turn may produce the pathologic changes reported by Hallpike and Cairns.

It is noteworthy that Mygind and Dederding,[7] in their report on the first large group of patients with Ménière's disease successfully relieved by medical treatment, based their management on the hypothesis presented by Parrisius and by Müller. Mygind and Dederding stated that Ménière's disease was not a disease *sui generis* but a typical reaction of a predisposed labyrinth to an almost infinite series of exogenous and endogenous influences which, however, have in common that they express themselves through increased capillary permeability.

All modern treatment of Ménière's disease has been based on the hypothesis first presented by Parrisius and by Müller.

THE APPROACH TO THE PATIENT

An important part of the treatment of Ménière's disease is the approach to the patient. Patients with this disorder are usually in an extremely tense and nervous state as a result of their syndrome. They frequently believe that their disease is the result of a serious intracranial lesion. They wish to tell their symptoms at great length. To gain the confidence of such a patient, the physician must be willing to obtain a careful and detailed history and make a careful physical examination. He should use all the necessary cochlear and labyrinthine tests, obtain roentgenograms of the head (including a special view to show the internal auditory meatus), examine the pupils and ocular fundi, and carry out at the very least a Romberg test and a test for corneal and facial anesthesia. When the physician has assured himself that the history and findings are consistent with the diagnosis of Ménière's disease and do not represent some condition such as vertiginous epilepsy, vertiginous migraine, thrombosis or embolism of the posterior cerebellar artery, an anxiety attack with hyperventilation, multiple sclerosis, platybasia, a lesion of the cerebellopontine angle, or a lesion of the cerebellum, he may then reassure the patient. The patient should be told that he does not have a dangerous intracranial condition but that the disorder is due to an interference with the blood supply to the labyrinth. He should be told also that since the disorder is of an inherited nature it cannot be "cured" because his in-

herent tendency to react to stress as he does cannot be eliminated but in most cases the disease itself can be greatly ameliorated by relatively simple medical means. However, the patient is less robust than some of his more fortunate acquaintances, and if he expects to control the disease, he must lead a more relaxed and less vigorous life than a normal person.

The definitive treatment of Ménière's disease can be logically divided into that of the acute attack, or crisis, and that of the persistent underlying condition.

THE ATTACK, OR CRISIS, OF MÉNIÈRE'S DISEASE

Because the reaction of the peripheral vascular bed (the arterioles and capillaries) is identical in Ménière's disease with that of Raynaud's disease, migraine, acrocyanosis, physical and immunologic allergy, and the vasomotor neuroses, the condition has been classified as a dysfunction of the autonomic nervous system and causally related to parasympathetic or cholinergic hyperactivity.

If this hypothesis is correct, those drugs stimulating the adrenergic or paralyzing the cholinergic nerves would be expected to be successful in controlling the condition. This has proved to be true in controlling the acute attack.

Atropine, a cholinergic antagonist, was early found to be useful in relieving the symptoms of an acute attack of Ménière's disease. One seventy-fifth of a grain (0.00086 gm.) given subcutaneously will usually be effective in terminating the crisis in from 15 to 30 minutes.

Of the drugs stimulating the adrenergic nerves, epinephrine is also active in relieving the acute attack. It may be given subcutaneously, but giving 5 to 10 minims (0.3 to 0.6 cc.) of a 1:1000 solution slowly intravenously, with termination of the injection as soon as definite improvement is evident, will ordinarily prove effective in relieving the crisis of the disorder in a relatively short time. In persons who have warning of an impending attack, ephedrine by mouth in the dosage of 1½ grains (0.1 gm.), repeated in 15 minutes if necessary, has also been found effective. Amphetamine by mouth in doses of 5 to 10 mg. has also been found useful.

Recently, injectable dimenhydrinate (Dramamine) has been found effective in relieving the acute attack. This is furnished in a 5 cc. vial, containing 250 mg. of dimenhydrinate in solution. This may be given directly by the intramuscular route or intravenously by a further dilution with 10 cc. of isotonic saline solution.

THE UNDERLYING CONDITION IN MÉNIÈRE'S DISEASE

In most patients with Ménière's disease endolymphatic hypertension is present. Occasionally a patient with this disorder will be seen with typical recurring attacks of severe vertigo and vomiting, on whom tests give no evidence of a cochlear or vestibular disturbance of this type. In these cases the assumption can be made that vasomotor ischemia affects the blood supply to the cristae or macula without involvement of the vessels supplying the striae vasculares. Since the problem without endolymphatic hypertension is one of controlling vasospasm, medical treatment in this type is usually very effective, the vasodilators alone often being capable of producing the desired result.

The first vasodilator used in the treatment of Ménière's disease was heat supplemented by massage, to improve vasomotor tone and correct the peripheral splanchnic balance. Physical medicine will still be found a helpful agent in relieving the symptoms of autonomic imbalance.

Papaverine appears to have been the next vasodilating agent used in this condition. Russek and Zohman [8] suggested a daily dose by mouth of from 4½ grains (0.29 gm.) to 12 to 18 grains (0.78 to 1.2 gm.), depending on the individual response to the drug.

Harris and Moore [9] suggested the use of nicotinic acid in Ménière's disease. This also has been found effective in relieving the condition. Nicotinic acid tablets of 100 mg. may be given by mouth, and the number increased at each dose until an effective flush is produced. The most effective time

for this medicine to be taken is after rising and before breakfast. It seems to be effective if taken once daily. Dosage should be continued from four to six weeks after the symptoms subside. On the return of symptoms, a "booster course" may be taken for a week. Intermittent dosage of this type may have to be continued for an indefinite period.

Histamine also has been used as a vasodilator. Unfortunately, the term "histamine desensitization" became current, and the idea became prevalent that a specific sensitivity to histamine might be relieved by gradually increasing the dosage. This led to the injection of small increasing doses of histamine, and this plan of administration has not proved effective. To be effective the intravenous drip method advised by Horton[10] should be used in administering histamine. He advised that 2.75 mg. of histamine phosphate diluted in 250 cc. of an isotonic solution of sodium chloride be administered by the drip method at rates ranging from 16 to 60 drops per minute, depending on the tolerance of the patient. He found that it was the rate of administration of the solution, and not primarily the amount of histamine given, which was of paramount importance. The rate of administration should be such that the patient has no headache and no discomfort whatever. The stomach should be protected by foods or antacids against increased gastric acidity. The injection is usually given daily for as long as it is found necessary to control the symptoms.

In Ménière's disease with endolymphatic hypertension or in endolymphatic hypertension without vertigo, in addition to releasing the arteriolar spasm, the problem of relieving extracellular (interstitial) fluid must be considered. It was stated previously that when the stria vascularis was involved an increased amount of endolymph with increased sodium and protein was secreted.

Smith and associates[11] found that in the endolymph of the normal guinea pig the concentration of potassium is 30 times that in the perilymph and the concentration of sodium is only $\frac{1}{10}$ as high as that in the perilymph or cerebrospinal fluid. They found endolymph and cerebrospinal fluid to be low in protein but the perilymph contained twice as much as either of the other fluids. It seems possible that in Ménière's disease there is a definite shift in the electrolyte content of the endolymph, with an increase in the ratio of sodium to potassium. This might affect the "positive D. C. potential" of the scala media, as demonstrated by von Békésy,[12] and interfere with the initiation of nerve impulses in the organ of Corti. These changes might play as great a part in the hearing loss in Ménière's disease as the squeezing of the organ of Corti produced by endolymphatic hypertension.

The slow shifts in the hearing evident in the early stages of Ménière's disease are quite different from, and not necessarily synchronous with, the sudden attacks of vertigo and nausea. The latter are considered due to vasospasm of the nutrient vessels to the cristae and macula, the former to the slower changes in the composition of the endolymph.

The loss of hearing in Ménière's disease may appear months to years before the advent of vertigo, appear at the time of the first attack, or not appear until a series of recurrent attacks of vertigo has occurred.

In treating Ménière's disease with endolymphatic hypertension the problem of an increased collection of the endolymph, which is, in fact, a collection of interstitial fluid or edema fluid, must be dealt with.

Schemm[13] stated that a volume increase in interstitial fluid can accumulate and exist only if its materials have been supplied and retained. For each kilogram of such edema fluid the materials consist of 10 gm. of an alkaline mixture of sodium salts and 1000 cc. of water as a solvent. The collection of alkaline interstitial fluid remains inertly and indefinitely maintained unless the bicarbonate fraction is used up by the metabolic acids or by ingested acid. Acidification incites the kidneys to balance the threat to the hydrogen-ion concentration of the body fluids by the elimination of neutral or acid sodium salts. Acidification mobilizes the sodium.

Schemm further said that it is necessary to decrease the ingestion of sodium and encourage its mobilization by giving a diet restricted in sodium and yielding a neutral or acid ash. The normal effect of the metabolic acids should be increased by the administration of acid drugs. The elimination of the mobilized sodium via the kidneys should be facilitated and the development of true cellular dehydration evaded by giving plain water in adequate amounts. Keith and his associates [14] have suggested the use of ammonium chloride in divided doses of from 6 to 9 gm. daily as probably the most effective diuretic in Ménière's disease, since it has a greater power to produce relative acidity of the tissue fluids than even the mineral acids.

Perlman and associates [15] were unable to confirm the findings of Mygind and Dederding [7] and Furstenberg and associates [16] in regard to the effect of sodium restriction in Ménière's disease. They studied 15 patients with Ménière's disease (6 of whom had bilateral involvement) in the hospital on programs that produced measured increase or decrease of levels of serum sodium. They were unable to note any consistent effects on vestibular or cochlear function in patients with a measured low serum sodium. Random fluctuation in hearing continued, and some acute severe attacks of vertigo were observed. Furthermore, no acute attacks of vertigo were seen in patients whose serum sodium had been sharply raised by the giving of cortisone, and no constant deterioration of cochlear function was produced. They felt that their work indicated that the present therapy directed at relieving the symptoms of Ménière's disease by changing the electrolyte composition of the labyrinthine fluids is probably ineffective.

Their results in patients with Ménière's disease are in sharp contrast to those obtained by Dederding [17] and by Furstenberg and his associates by decreasing, and by increasing, the intake of sodium in patients with Ménière's disease. Furthermore, in our own hands 200 mg. per day of cortisone given to one patient with long-standing Ménière's disease without fluctuation of hearing produced a decrease in hearing of 5 db. after three days; this loss of ability to hear was maintained as long as the cortisone was administered (a two-week period). After administration of cortisone was stopped, the previous level of hearing was regained in three days.

It is possible to conceive that the lack of effective results reported by Perlman and associates was influenced by the large number of patients with bilateral Ménière's disease included in their study. It is well known that patients with bilateral Ménière's disease give no favorable response to any form of therapy. It seems then that the lack of results reported by these authors requires further confirmation.

I recommend for my patients a modification of the diet proposed by Schemm, which is not only low in sodium but also has an acid to neutral ash; the intake of pure water is not restricted. This diet, known as the low-sodium acid or neutral ash diet, modified from Schemm,[13] is as follows:

Maximal Intake in 24 Hr.	
Food	**Amount**
Milk	1 pt.
Vegetables	2 servings
Fruit	2 servings except for prunes, plums, cranberries

Minimal Intake in 24 Hr.	
Food	**Amount**
Eggs	2
Meat, fowl, fish	2 servings
Cereal grains	Equivalent of 6 slices of bread
Prunes, plums, cranberries	As desired

1. Avoid soda, such as baking soda, soda biscuits, soft drinks, and water containing soda.
2. Avoid adding salt to food in addition to the seasoning added in the kitchen.
3. Avoid foods prepared with salt, such as sauerkraut, salt codfish, herring, and the like.
4. Avoid salty broths.

In addition, 6 to 9 gm. of ammonium chloride in enteric-coated capsules in divided doses is given daily for three days, omitted for two days, and then repeated.

Williams and associates[10] found that women with Ménière's disease who were also subject to the syndrome of premenstrual edema tended to have exacerbations of Ménière's disease in the premenstrual stage of fluid retention. They found that the administration of small doses of thyroid and estrogen tended to counteract this tendency.

It also seems that sedatives to control the anxiety tension state present in many patients with Ménière's disease are helpful in alleviating the symptoms. It is suggested that phenobarbital, 1 grain (0.065 gm.) given twice daily, or N-methylethylphenylmalonylurea (Mebaral) should be used.

It has been found more effective to combine the several methods of treatment—the vasodilators to "unlock" the arteriolar spasm, the acid-ash low-sodium diet to decrease endolymphatic hypertension, endocrine therapy when indicated, and sedatives to relieve tension and anxiety—than to give one after another. It has been estimated by various authors that the symptoms of Ménière's disease can be alleviated in about 75% of patients by medical treatment.

SUMMARY

While it is admittedly difficult to evaluate the effect of medical treatment in a group of persons in whom, as in those who have Ménière's disease, the disorder depends on an inherited constitutional tendency to react to stress in an abnormal manner, it seems definitely possible to diminish and control the symptoms of these patients by a proper medical regimen. However, too much should not be expected from medical therapy. Obviously, the tendency to react cannot be abolished by any therapy. Therefore, the expressions of the disorder may be controlled but not abolished.

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Nasopharyngeal Lymphoid Tissue and Its Relationship to Hearing

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It is the purpose of this paper to present some observations on the relationship of ear disease and nasopharyngeal lymphoid tissue. Interest in pharyngeal lymphoid tissue occurred very early in the history of medicine. Hippocrates referred to swellings and inflammations of the throat in 400 B. C. Celsus described a method for removing the tonsils in 50 A. D. The word adenoid first appeared in the literature during the Roman domination of medicine. Alubucasis published what is perhaps the first account of the indications for tonsillectomy, about 1100 A. D.[1] Numerous reports on this subject have been published, but many questions concerning this problem still remain unanswered.

Clinical evidence has long supported the assumption that diseases of the Eustachian tube and middle ear are frequently related to changes of the lymphoid tissue of the nasopharynx. The anatomical continuity of the structures of the Eustachian tube and the nasopharynx is well established. As Robison[2] states, "The pharyngeal orifice of the Eustachian tube shares in the inflammatory reaction of the nasopharyngeal tissue." The major portion of the problem of correcting or preventing middle-ear disease and hearing loss in children resolves itself into the control of the lymphoid tissue of the nasopharynx.

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The removal of the adenoids and tonsils does not entirely answer the problem. Adenoidectomy for the relief of recurrent otitis media in children 3 years of age or under is very effective. This is especially true in those cases where enlarged adenoids obstruct the airway. A similar opinion was expressed by Kaiser[1] in 1932. On the basis of a 10-year study of 4400 children, he concluded that between the ages of 1 and 3 years adenoidectomy offered considerable protection against recurrent attacks of otitis media. After the age of 5 years this procedure had less value in preventing acute ear disease.

In chronic disease of the middle ear, such as secretory otitis media, the number of cases permanently cured by the removal of the tonsils and adenoids is small. In a study of 100 children from whom the tonsils and adenoids were removed for chronic secretory otitis media, 24 remained free of symptoms for two years or longer without additional therapy. Hoople and Blaisdell[3] seem to share this view in their report of 804 cases of secretory otitis media, in which they attributed only 50 cases to diseased tonsils and adenoids. The value of tonsillectomy and adenoidectomy for the relief of this condition may be misleading because initial improvement following surgery is common. For a short period of time the middle ears may become free of fluid and properly ventilated, but recurrence of signs and symptoms is seen in the majority of cases from 2 to 21 months after surgery.

Hypertrophy of the remaining lymphoid tissue in the nasopharynx frequently occurs in those cases which have not been improved by tonsillectomy and adenoidectomy. This

hypertrophy is manifested by enlargement of the lateral pharyngeal bands and the appearance of discrete raised nodules over the surface of the oral and nasopharynx. Meltzer [4] has shown that this hypertrophy is due to hyperplastic lymphoid tissue. This tissue may react to infection and allergic stimulation the same as tonsil and adenoid tissue. If this hypertrophy becomes excessive, two methods for reducing the amount of this tissue are commonly employed: surgery and irradiation. If the mass is of sufficient size and in an excisable location, much tissue can be removed surgically. Smaller remnants of tissue and adhesions in the Rosenmüller fossae may be removed by finger manipulation or punch forceps. Regardless of the methods used, it is virtually impossible to surgically remove all the lymphoid tissue in the nasopharynx. Too vigorous surgery can result in excessive scarring and even atresia of the nasopharynx.

Because of the sensitivity of lymphoid tissue to irradiation, x-ray or radium is used to reduce this hyperplastic tissue. X-ray is preferred because of the larger area covered by the radiation, the greater accuracy of dosage, and the increased safety offered by the experienced radiologist. In addition, preliminary studies indicate a higher percentage of good results are obtained by directing the tube so that the x-rays pass through the middle ear, mastoid, Eustachian tube, and the lateral pharyngeal wall. It is possible that some of the edema often present in the Eustachian tube in certain ear conditions may be reduced by this method of exposure.

Using the present accepted dosage of x-ray or radium, one cannot expect to see a great reduction of the amount of tissue in the nasopharynx following this form of therapy. In a series of cases reported in 1949 [5] in which the adenoids of children were exposed to the radium applicator prior to removal, no gross or microscopic changes due to radiation could be demonstrated in those cases operated on three weeks after the last irradiation. One to three exposures of 12 minutes each were applied to each side of the adenoid mass. Removal was performed 18 hours to 6 weeks following the last exposure. The only true microscopic evidence of radiation effect occurred in the 18-hour specimens. Meltzer [4] states that in his years of experience with radiation he has never seen a lymphoid mass resolve from its use. Schenck [6] feels that the beneficial result from radiation is reopening of lymph channels and resumption of normal lymph flow, rather than any diminution of the lymph follicle.

A study of 80 children with chronic Eustachian tubal block and nasopharyngeal lymphoid hyperplasia was made to test the efficiency of secondary adenoidectomy and radiation therapy. In this group, surgery and radiation were combined in the majority of cases. Forty-one patients remained well after 2 years; 39 showed no improvement or had recurrence of symptoms within 21 months. In the 39 cases which failed, the greatest number had recurrence during the second month.

The presence of normal lymphoid tissue in the pharynx does not cause ear disease. Some additional factor such as allergy, infection, or both must be present before ear pathology occurs.

Allergy is an important factor in recurrent ear disease. Tissues of the nasopharynx, Eustachian tube, and middle ear may become shock areas to offending allergens. Children with secretory otitis media secondary to Eustachian tubal closure from allergic edema are commonly seen. In addition, allergic edema of the nasopharynx increases the patient's susceptibility to secondary infection. This is especially true of very young children.

Infection plays a major role in ear disease. Frequently a history of repeated attacks of otitis media associated with acute infection of the tonsils and adenoids is obtained. Apparent recovery may occur after administration of antibiotics, but the performance may be repeated after three to five weeks. Between acute attacks it is not uncommon to find the middle ears of these children filled

with fluid, either an exudate or a transudate, especially if the drum has not been incised for the acute infection. Rapid recovery from acute otitis media following the administration of adequate doses of antibiotics may discourage the formation of local immunity and increase the susceptibility to repeated attacks. Attenuation of the organisms by inadequate or improper antibiotics may result in the tissue of the nasopharynx becoming sensitized to the offending bacteria. Drinker [7] has shown that nasopharyngeal lymphoid tissue need not be overwhelmed by an acute infection to develop edema and hyperplasia. Such changes can occur when only a few bacteria and viruses of low virulence are present. Meltzer [4] concluded from this work that tissue about the torus and in the Rosenmüller fossae becomes a shock area as the result of bacteria in the adenoids. Removing the tonsils and adenoids may reduce the amount of tissue in the nasopharynx capable of harboring organisms but may not improve local immunity or reduce the sensitivity of the adjacent tissue. Neither is the immunity improved or the sensitivity controlled by antibiotics, regardless of the type or amount used.

If a sensitivity exists to foods, inhalants, or bacteria, some attempt must be made to control these offending allergens. Antihistaminics have been helpful in mild cases. Desensitization or elimination diets are required in the more persistent cases. It has long been my practice to use stock respiratory vaccine in combination with dust injections. More recently I have found that autogenous vaccines give better results. These preparations are made from cultures taken directly from the nasopharynx and given weekly in doses below the level of systemic reaction. The change to autogenous vaccines was prompted by the work of McCollough * and Beauregard,† who studied cultures of the nasopharynx of 198 children with persistent or recurrent ear disease. Hemolytic Staphylococcus was found to be the predominating organism in the majority of cases. Most stock vaccines contain more hemolytic Streptococcus than Staphylococcus. The predominance of hemolytic Streptococcus in stock vaccine may be a reflection of the work of Polvogt and Crowe [8] reported in 1929. In their series of 100 cases in which the tonsils and adenoids were cultured, the hemolytic Streptococcus was found to be the predominating organism in 81%. These findings suggest that a change in the bacterial flora of the nasopharynx has occurred in children in recent years.

Regardless of the presently accepted procedures used to control ear disease secondary to pharyngeal lymphoid changes, there will remain a small group which do not respond to treatment. Many of these are children whose allergies cannot be controlled. Perhaps some of these cases are due to a virus, such as recently reported by Huebner, Rowe, Ward, Parrot, and Bell. These men have isolated 143 different strains of virus from adenoid tissue, some of which are capable of producing disease in the nasopharynx. At present there is no specific treatment for these viruses.

Although pathologic changes of the lymphoid tissue of the pharynx have been apparent to the medical profession for centuries, the etiology is not yet fully understood. Many factors, such as the effect of environment, diet, hygiene, and endocrine dysfunction on this tissue, have not been completely explored. More research will be necessary before this complex problem is resolved.

CONCLUSIONS

1. Simple tonsillectomy and adenoidectomy is frequently insufficient to control ear conditions secondary to pharyngeal lymphoid disease.

2. Secondary surgical removal of hyperplastic lymphoid tissue in the nasopharynx is frequently necessary to resolve ear conditions.

3. Irradiation by x-ray is effective in reducing ear disease secondary to lymphoid changes in the nasopharynx.

* McCollough, T. B.: Personal communication to the author.

† Beauregard, J. F.: Unpublished personal report.

4. Allergy is frequently associated with recurrent ear disease in children. Allergic management is necessary in the majority of cases before control of the disease is accomplished.

5. The antibiotics have not been effective in controlling recurrent ear disease. Better results are obtained from the use of autogenous vaccines.

6. Other factors, such as environment, diet, hygiene, and endocrine disturbance, must be considered.

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Hypothyroidism as a Cause of Headache

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Wolff [1] said that pain is certainly the commonest complaint with which the physician must deal, and headache is probably the commonest pain. The headaches which we see as physicians can be divided into major headaches and those which might be termed minor headaches. It is with the latter type that this paper deals, and, thus, major headaches can be eliminated from the discussion.

The minor headaches are frequently passed over by the physician and given a little thought, then are promptly dismissed or are treated as a sinus headache by some type of nasal drops or antibiotic. The purpose of this paper is to point out that there are many minor headaches which are of considerable concern to the patient and to report on the observations made on 118 patients with this type of headache and the response obtained in their treatment with thyroid after a diagnosis was made of headache due to a low metabolism.

The diagnosis of any headache depends on a detailed history of the patient as a whole. In addition to the detailed history of the headache, including the location of the headache, the frequency, the severity, the type, duration, and the associated symptoms, many factors which do not directly relate to the headache, such as possible causes of a tension state, a review of the psychic history, and problems related to the endocrine and hormonal systems, must be investigated. In such a history one will find that the headache usually is not severe. It may be variously located about the head in the distribution of some of the cranial nerves or related to the nose or the paranasal sinuses, or it may be an ill-defined aching any place in the head.

After the history is obtained, an examination of the ears, nose, and throat must be carried out and x-rays taken when indicated. These will be without evidence of pathology, except occasionally one will find a bluish discoloration of the nasal mucosa which is frequently attributed to a nasal allergy. The laboratory work consists of basal metabolic rate determinations, and, in these particular patients, they may be normal or just slightly subnormal. Blood cholesterol determinations usually will be slightly elevated, and a protein-bound iodine determination, if possible, would be confirmatory. The facilities for such determinations have not been available in my locality until quite recently; therefore, it has been impossible to check this determination in the present group of patients.

There are other points of diagnostic interest that have been of assistance in arriving at the diagnosis of mild hypothyroidism. One of these is a subnormal afternoon temperature as checked on several occasions. Another, as pointed out to me by my father,* is the presence of a varying but definite infraorbital edema. A third factor is a resting pulse rate which is slightly low. One must instruct the patients to take their own pulse rate before arising in the morning for three days and then average these. Also, this resting pulse rate observation helps in determining the adequacy of treatment.

Referring to the basal metabolic rate determination, Bryant [2] pointed out, "By the nature of the test an error can occur only in a positive direction; hence, a minus

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* Jones, A. C.: Personal communication to the author.

six reading may actually represent a minus fourteen or fifteen, whereas the opposite can never be true." It is often helpful in determining the actual basal metabolic rate to repeat the test on three separate occasions to overcome some of the patient's fear of the examination and, thus, to arrive at a more accurate basal metabolic rate determination. It should be emphasized that the metabolism is borderline low in these patients with headache as the result of a low basal metabolic rate. If these patients had true myxedema, the diagnosis of hypothyroidism would be simple.

The mechanism of the development of pain in these persons with mild hypothyroidism is not definitely established. It is recognized that the fault may lie in one of three levels as far as the endocrine balance is concerned. First, there is the little-known primary mediator of the pituitary which stimulates the pituitary gland. Second, the pituitary then produces the thyrotropic hormone which stimulates the thyroid. Third, the thyroid produces its hormone which, in turn, has some effect upon the pituitary and the remainder of the endocrine system. This interplay has a direct effect upon the fluid balance of the body as a whole, and when the thyroid is underactive a transudate problem will exist, according to Hilger.[3] Hilger believed that this transudate then causes the headache. The transudate is produced by little-known causes. It may be due to changes in the capillary epithelium, shifts in the electrolyte balance between the vascular and the extravascular spaces, or tissue changes such as those which increase the subcutaneous mucopolysaccharides of myxedema. When this transudate is produced about the vasa vasorum of the pain-sensitive vascular network of the head, pain will result in one or more branches of that vascular network. When transudate occurs in the connective tissue of the nose and paranasal sinuses, the pain will be referred to these structures and frequently will be confused with a sinus infection. There may also be a relationship between a hypothyroid state and the ground substance of the connective tissue mentioned by Rawlins.[4] However, since this theory enters into the field of other functions of the pituitary and the problems related to corticotropin and to cortisone, it is outside the scope of this paper.

At this point, I would like to relate the history and treatment of one patient of the present group of 118 who fairly well typifies this problem and its therapy.

REPORT OF A CASE

A 36-year-old white farmer was referred to our clinic because of a "sinus" headache. He was a thin, somewhat nervous-appearing man who was otherwise in good health. The history revealed that he had had a persistent frontal headache for several years. This headache was never severe and was not incapacitating; however, he stated that he felt run down most of the time. Previous treatment had consisted of various types of nose drops, and on several occasions he had received antibiotic therapy for short periods, all without relief. It was his opinion that the headache was the cause of his being run down.

Examination of the ears, nose, and throat showed no signs of any pathology in these organs, and, while there was no evidence of sinus infection, x-rays were taken to eliminate the possibility of any pathology there. The x-rays of the sinuses were within the realm of normalcy. This man showed a slight puffiness under his eyes. At the time he was originally seen his temperature was 98.6 F. He was referred to the laboratory for basal metabolic rate and blood cholesterol determinations, which were done the following morning. The basal metabolic rate was recorded as +3%; the blood cholesterol was recorded as 305 mg. per 100 ml. In spite of the relatively normal metabolism recording, he was placed on thyroid extract, 1 grain (60 mg.) a day, and was advised to continue this medication for one month.

At the end of that month he returned and reported no improvement in his headache. A repeat basal metabolic rate determination was recorded as —15%. He was placed on 2 grains (0.12 gm.) of thyroid daily. This brought his basal metabolic rate up to —10%; however, he stated he still had his headache and was quite discouraged about this type of treatment. His dosage of thyroid extract was increased to 4 grains (0.25 gm.) a day, and at the end of three weeks he stated that he was completely free of his pain and for the first time in years he felt like getting out and working. At this time his basal metabolic rate determination was +2%.

This man was not seen again for two years. When he returned with a recurrence of his head-

ache, his subjective history revealed that he had continued taking 4 grains of thyroid daily for eight months, and then, because he was feeling so well, he felt that he could discontinue the medication. It was not until some three or four months after he discontinued the medication that he noted onset of his headache again and gradually noticed that he was not feeling quite up to working. Thyroid medication was reinstituted and brought up to 4 grains of thyroid a day, and he was again free of his headache.

COMMENT

In this particular patient it would have been better to have repeated the basal metabolic rate determination on three occasions prior to instituting treatment, and certainly it would have been better had he not discontinued his medication after having been free of his headache for several months.

In our group of patients were some who were relieved of their headache on as little as ½ grain (30 mg.) of thyroid extract a day, while a few have required as much as 8 grains (0.53 gm.) of thyroid extract a day. It must be emphasized that, after the proper dosage of thyroid extract has been determined, the patient should be strongly advised that this in no way is a cure of the condition and he must continue to take the thyroid daily in order to remain free of the headache.

There were some patients in this group of 118 in whom we could find no evidence of a borderline hypothyroid condition but who were empirically placed on thyroid extract for one to two months and were similarly relieved of their headache. Occasionally, patients will complain of feeling jittery when they take thyroid extract. These patients should be advised to check their morning pulse rate prior to getting out of bed. It is recommended that the resting pulse rate be kept below 90 per minute and that the intake of thyroid extract be adjusted so that the resting pulse remains below this level.

In conclusion I would like to point out that in our clinic we have ophthalmologists and otolaryngologists but no endocrinologist. If in any way our treatment of these patients has not agreed with the theories of the endocrinologists, we submit that we are trying to relieve the patients of their headaches and believe that we have benefited many and, as far as we have been able to tell, we have harmed none with this type of treatment.

SUMMARY

Headaches can be caused by a low metabolic rate, and these headaches can be relieved by administration of thyroid extract. It is essential that prior to treatment a detailed history be obtained to rule out any other cause of the headache and that a physical examination be performed to determine whether any other pathology exists. The only contributory physical findings are an infraorbital edema, subnormal afternoon temperature, and a slow resting pulse rate. Repeated basal metabolic rate determinations are necessary to find the actual basal metabolic rate of the given patient, and during treatment with thyroid extract further basal metabolic rate determinations must be made to assure oneself that the patient is receiving adequate treatment. In the past six years 118 patients were so treated for relief of their headache.

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Radical Surgery on Os Temporale

A Study Based on Newer Concept of Pathological Changes in Infection of Temporal Bone

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During the past half century, the concept of otitis media has undergone a gradual evolution. With a better understanding of the basic pathological changes that develop in temporal bones, the operative procedures dealing with infectious processes have also shown a slow but steady evolution.

In 1904 on the basis of the autopsy examination of Bezold and Politzer, Scheibe [1] published the following opinion: In the cases of otitis media in which one elicits tenderness over the mastoid process, one may be justified in considering that there exists an extensive inflammation of the pneumatic cells as well as in the tympanic cavity. In 1912, Alexander [2] wrote that the time relation varied between the onset of mastoiditis and that of acute purulent otitis media. In cases of the fulminant type of purulent otitis media in which suppurative changes had existed for only a few days, he noted the surprising fact that the mastoid process almost always contained pus.

This is an important report. However, because Alexander limited this condition to "fulminant otitis media," the general impression imparted was that in cases of ordinary otitis media these findings would be different. Denker and Albrecht [3] (1925) wrote that in acute suppurative otitis media the membrane of the tympanic cavity and the mastoid cells were involved at the same time in the majority of cases. But the descriptions under the paragraph on acute otitis media and mastoiditis do not conform with this statement, and no data were reported by them which would support their assertion. They did not overthrow the concept held by otologists that the mastoiditis is a complication of otitis media.

Mayer in 1927 reported the histopathological findings in the mastoid cells of patients who died on the third and fourth days of the illness, respectively. According to this report, there was an extensive inflammation in the mastoid areas.

In 1928, he [4] stated that the acute mastoiditis is not a result of acute otitis media but is a part of the disease. Lange,[5] Krainz,[6] and Schlittler [7] described histologic evidence of extensive inflammatory changes in the mastoid cells in those who died in the first four days of an illness. Portmann [8] shared a similar view. He named the condition "mastoid reaction."

In 1931, Linck * stated that the conception of otitis media should be changed, since in acute otitis media the infection also occurs in the mastoid and pyramid. On the basis of this revised conception, he brought out a new classification of otitis media.

Since 1925,[10] I have repeatedly observed that (1) even in the absence of classical signs of acute mastoiditis at times the otorrhea was definitely out of proportion to the size of the middle ear cavity; (2) this led me to feel that suppurative lesions in acute otitis media must develop at deeper and much more widely scattered areas in the

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* Reference 9. Other articles on the subject by Linck appeared in the same journal, Vol. 145, 1938.

temporal bone; (3) if otitis media were a simple tympanitis, as we were led to believe, then we should be able to bring about its resolution by a simple paracentesis, but (4) some of these acute cases went on to develop chronic lesions. On the basis of these observations, I concluded that in every case of acute otitis media the mastoid cells were also involved early in the inflammatory process. Though lacking in animal experimentations, my concept of the pathological changes in temporal bone has been greatly clarified by subsequent clinical observation.

In 1935, I had the opportunity of operating on three cases of otitis media on the second or third day after the onset of the infection. Notwithstanding the fact that these cases had not exhibited any symptoms of mastoiditis, a purulent inflammation was found in the mastoid cells. Subsequently, I encountered numerous similar cases.

The point I wish to bring out is that, as I had surmised, there are many cases in which the mastoid cells are affected at an early stage of acute otitis media.

In 1938, Marx [11] described the relationship between otitis media and mastoiditis, but it is difficult to grasp his meaning because of lack of consistency. For instance, he stated, "In the case of exudative inflammation of the tympanic cavity, the mastoid cells are always involved, but this is found only in a hematogenic infection, and in cases of tubal infection, the tympanic cavity is first affected and subsequently the infection extends to the mastoid cells."

This view is nothing new, but in subsequent pages he wrote: "In almost all cases infection extends to the mastoid cells through the mucous membrane. If this is called mastoiditis, any kind of otitis media is related to mastoiditis."

Perhaps he took the histopathological definition of mastoiditis too seriously, and he must have considered the fact that a condition should not be called mastoiditis if inflammation extends to mucous membrane only.

There are many definitions of mastoiditis, but the focal point is based on the histopathological findings, and from the clinical standpoint one cannot definitely establish the diagnosis of the disease. However, the diagnosis of mastoiditis is usually made clinically, and it is not clear-cut.

A more important matter is to know when the inflammation does attack the mastoid cells. It is clear in the literature that inflammation is recognized macroscopically only in the mucous membrane, but on histologic investigation one finds concomitant bony changes. Thus, there is no essential difference whether one calls the condition mastoiditis, mastoid reaction, or mastoidismus.

The fact is that inflammation which affects the mastoid cells or pyramid at an early stage of otitis media explains the pathology of otitis media and the mechanism that causes endocranial complications before clinical diagnosis of mastoiditis or petrositis is made.

PETROSITIS

Oda,[12] Eagleton,† and Lempert [15] added much to the present knowledge of petrositis. Based on their clinical observations, Sawatari and Niho [16] introduced a new term, osteitis temporalis, to describe the infectious process in the temporal bone. M. Niho and his associates [17] demonstrated histologically that bony necrosis occurs in acute otitis media. Isono [18] observed similar changes in 60% of 500 cases of chronically draining ears upon which he operated. Miyao [19] recorded also that diffuse changes in temporal bone develop in chronic catarrhal otitis media.

However, these views are not shared by all our otologic colleagues. Both Torii and Goto regard acute otitis media as a separate entity from acute mastoiditis.

In 1946, M. Niho ‡ proposed the so-called radical os temporale operation. In essence, it is a combination of various known pyramid operations (Streit, Yamashita, Voss, and

† References 13 and 14.

‡ References 20 and 21.

Fig. 1.—Malleus. Macular absorption.

Ramadier) for complete exenteration of pneumatic cells.

Upon surgical revision in 10 patients who continued to have persistent otorrhea after conventional radical mastoidectomies performed by other otologists, in most instances I found perilabyrinthine cells which were incompletely capped. The most neglected areas were in the anterosuperior cells of the pyramid, in the hypotympanum, and in the peritubal and anteroinferior cells, in the order named.

Fig. 2.—Peritubal area. Scattered necrosis of the bony structures (macular absorption and halisteresis).

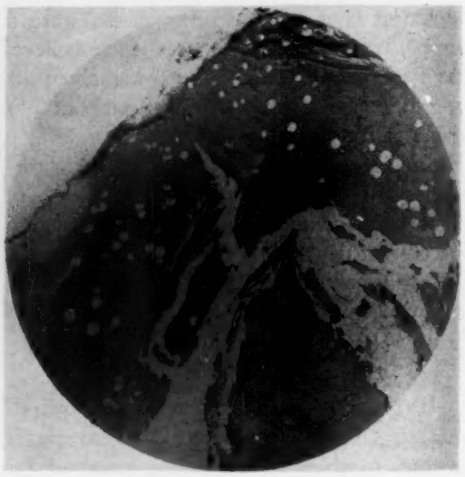

Fig. 3.—Petrous portion. Reticular and porous changes following bony absorption.

Histologic examination of the bone removed indicated destructive changes proportionate to the extent of the infection. However, bony changes in these sections were not as extensive or severe as those observed in acute otitis.

The most commonly observed bony changes are halisteresis and loss of nuclei of the bony cells. No amount of antibiotics can bring about complete healing in the presence of bone necrosis. The changes were oftenest observed in the petrous pyramid and the cells

Fig. 4.—Peritubal area. Extensive bony necrosis and absence of bony cells.

Fig. 5.—Perilabyrinth. Diffuse absence of bony cells.

around the Eustachian tube. Except by the surgical removal of these infected cells, complete cure is not obtainable. Where complete epithelization failed to occur after radical or modified radical mastoidectomy, there were invariably microscopic bone absorption and disappearance of bone cells.

I share the viewpoint that acute and chronic otitis media are osteitis temporalis and that mastoiditis and petrositis are not a complication but rather an integral phase of the extensive changes in otitis media. Thus, the surgical procedure should conform with this newer concept based on the changes observed in the entire temporal bone. Each case must be dealt with according to the extent of the lesion.

Lempert described minutely his surgical procedure on the tympanum, carotid wall, tensor muscles, and hypotympanum by curettage. He failed to discuss the problem of lesions in the petrous portion. If the result of curettage of the temporal bone is not satisfactory, it is usually because of failure to pay strictest attention to each area so emphasized by Lempert in his excellent presentation.

CONCLUSION

In almost all cases of acute purulent otitis media, the inflammation extends not only to the tympanic cavity but also into the mastoid process and pyramid. This finding can be applied in chronic otitis media.

Mastoiditis and petrositis are to be considered a part of the extensive pathological change, rather than a complication, of otitis media. The surgical operation for treatment of otitis media should have all the parts of the temporal bone as an objective. Chemotherapy cannot cure the bony changes of chronic otitis media.

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Thrombocytopenic Purpura Due to Quinidine

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Werlhof in 1735 was the first to describe a symptom complex of hemorrhagic purpura. The essential type of this disease is sometimes referred to as Werlhof's disease.

Thrombocytopenic purpura is characterized by multiple hemorrhages into the skin and mucous membranes. One of the earliest indications of hemorrhage may be epistaxis, and the otolaryngologist may be the first to suspect the diagnosis and be instrumental in the institution of early treatment.

Thrombocytopenic purpura is characterized by prolonged bleeding time, poor clot retraction, an increase in capillary fragility, and a decrease in circulating platelets. It may be classified as (1) the essential, or primary, type and (2) the symptomatic, or secondary, type. In 1946 Dameshek and Miller suggested that the essential type was due to a splenic factor acting to prevent the formation of platelets. The symptomatic type was found with hypoplastic bone marrow. This may be due to a nutritional deficiency or to bone-marrow suppression. A bone-marrow aspiration will serve to differentiate any blood dyscrasias. Symptomatic thrombocytopenia may also occur because of toxic substances acting on the megakaryocytes. This may occur in infection, chemical poisoning, drug and food allergy, and irradiation. The present-day trend is toward looking upon secondary thrombocytopenia as a reaction occurring in many different infectious processes and with the administration of various drugs. With the removal of the causative factor, the process usually regresses.

In the toxic type of thrombocytopenia it is important to remove the causative factor. It is especially important to recognize the drugs that can be causative agents.

The first case of thrombocytopenic purpura due to quinidine was reported in 1941. It was suggested at that time that the mechanism may be one of anaphylactoid reaction, with the platelets as the shock tissue. Not only is there marked inhibition of platelet formation but there is an increase of platelet destruction. There have been 15 cases of thrombocytopenic purpura due to quinidine previously reported. These were reviewed and summarized by Weisfuse and associates in 1954. The symptoms that occurred in the previously reported cases were the following: weakness, epigastric distress, chills and fever, pruritus, petechia, and ecchymosis. Bleeding from the gums, epistaxis, hematuria, and oral ulceration also occurred. This review also gave the following information: Thirteen of the patients were females and one was a male, ranging from 26 to 76 years of age. Many of the cases showed no reaction to one dose of quinidine but reacted after readministration of the drug. A peripheral eosinophilia was sometimes present.

The case described in this paper presents a typical picture of thrombocytopenic purpura caused by quinidine.

REPORT OF A CASE

History: A 73-year-old white woman was admitted to Sinai on Jan. 7, 1955, on the medical service of Dr. E. Kallins. The admission diagnosis was (1) arteriosclerotic heart disease, (2) upper respiratory infection, and (3) pneumonitis. Digitoxin intoxication was to be ruled out. On admission the temperature was 104 F. Tetracycline (Achromycin) was started. Digitoxin was discontinued,

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and the patient was started on quinidine at 10 p. m. on Jan. 7. The temperature fell to normal in 48 hours. On Jan. 11 epistaxis started. Several bleeding points on the septum were cauterized. On Jan. 12 the epistaxis became severer and the patient developed hematuria, melena, diffuse skin petechiae, and bullous lesions of the mouth. The bullae were tense, filled with blood, dark purple in color, 2 cm. in diameter, and left ulcerations on breaking.

Treatment and course: All drugs were discontinued except quinidine. Vitamin K oxide emulsion (Mephyton) was given. After 12 hours all bleeding was more copious and petechiae more numerous. There were no platelets present on Jan. 12. The quinidine was discontinued. Six hours after quinidine was stopped the bullae were markedly diminished in size, the epistaxis was much less severe, and the petechiae had disappeared. The patient was started on cortisone, 75 mg. four times a day. All signs of bleeding disappeared, and the platelet count rose to 26,000 in 24 hours and on Jan. 20 was 510,000.

COMMENT

This case illustrates the importance of a complete blood work-up. What started as a simple epistaxis in an old person was a condition which may have proved fatal. An unusual cause of toxic thrombocytopenia, quinidine sensitivity, was the causative factor in this case. In the cases previously described, cortisone was used in only one instance. Cortisone should be used as an adjunct to discontinuing the drug to insure more rapid recovery.

CONCLUSIONS

1. In epistaxis cases blood counts with prothrombin determinations and platelet counts should be a routine step in the treatment.

2. In thrombocytopenic purpura cortisone brings a dramatic relief of symptoms.

3. Quinidine administration in some persons may cause a thrombocytopenia, which, if not recognized, may prove rapidly fatal.

2 E. Read St.

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Vocal Rehabilitation of Paralytic Dysphonia

II. Acoustic Analysis of Vocal Function

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INTRODUCTION

Persons with a hoarse voice are frequently referred to as having a "voice problem." While this is true in the broadest sense, it is obviously not enough to describe any particular vocal disability. It means about as much as if an eye specialist were to say that he is treating patients with "seeing problems," or if an audiologist were to write about the "hearing problems" seen in his hearing clinic without giving any of the elaborate and detailed findings commonly encountered in audiologic publications.

The great progress of medical science in this century is partly due to the rapidly advancing methods of analysis and description of physiologic functions. The same is true with regard to vocal function, and the medical specialty of voice therapy or phoniatrics has successfully kept pace during the first century of its existence with the progress of other medical sciences. Incidentally, the name of the phoniatric specialty is quite old, too. It was apparently first used by Sir Morell Mackenzie. One of his letters, dated Feb. 14, 1887, and published by Delavan in 1933, shows that he called himself "Phoniatros" and used this term as a cable address.

When dealing with vocal function, it is furthermore insufficient to state merely that the voice is normal, or disturbed, or totally lost. Just as the eye specialist would not be satisfied in saying only that a patient has good, poor, or no vision, the phoniatrician also needs more distinctions than euphonia, dysphonia, and aphonia. Parallels to further classifications may be seen in the accepted methods of evaluating auditory function. Audiometry has offered a large number of tests which can accurately and objectively describe the type, degree, course, and social significance of any specific impairment of aural function.

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CLASSIFICATION OF VOCAL DISORDERS

When a patient is hoarse, one first wants to know the cause of his trouble. Laryngologic and/or phoniatric examination provides this information. The "voice problem" is now subdivided into the various types of dysphonia. Acute or chronic inflammation of the vocal cords produces the well-known hoarseness as a symptom of these conditions which is designated inflammatory dysphonia. When a neoplasm, such as a polyp, fibroma, or carcinoma, is found, the hoarseness is called neoplastic dysphonia which again is symptomatic of the underlying disease. The hoarseness due to paralysis of any or all of the laryngeal nerves is paralytic dysphonia with the subdivisions according to the affected nerves. Traumatic dysphonia follows the various types of laryngeal injury and their sequelae. Thus, it goes on through the long list of endocrine, developmental, congenital, psychogenic, habitual, and other types of dysphonia whose causes have to be ascertained by the thorough and comprehensive studies now customary in all medical specialties.

Returning to paralytic dysphonia as a symptom of laryngeal paralysis, it can be said that this particular type of vocal disability is now fairly well clarified. Laryngeal paralysis has always been a fascinating problem to many laryngologists, so that there is now available an enormous bibliography on this subject. However, it was

only in recent years that the various aspects of laryngoscopic and vocal symptomatology of laryngeal paralysis were better understood. As the discussion of these anatomic and pathologic problems does not pertain to the subject of this paper, it will be presented later. Suffice it to say that the degree of vocal disturbance due to laryngeal paralysis depends mostly upon the degree of glottal closure and, thus, on the particular type of paralysis.

OUTLINE OF FUNCTIONAL EXAMINATION

Every analysis of a physiologic function must rely on the physiologic research in that particular field. It follows that an accurate description of vocal problems cannot be undertaken without a minimal knowledge of voice physiology. However, once this information has been acquired, it is easy to describe the various qualities of human voices in internationally accepted terms. Moses stressed in 1948 that such a procedure would be important for anyone who deals with laryngologic problems and who aims to restore or improve laryngeal function. It is not enough to say that as a result of a given surgical procedure the voice was better or clearer; such vague descriptions may mean something entirely different to different observers. For similar reasons, orthopedic surgeons evaluate their successes by various physical measurements such as degrees of movement, and otologists compare their postoperative results by quoting decibel differences of hearing acuity, percentages of speech intelligibility, or even indices of social adequacy.

The procedure of vocal analysis does not require much equipment. A fairly quiet office room without disturbance by telephones or typewriters offers enough privacy. For the recognition of musical qualities such as pitch, range, and registers, one needs some standardized sound sources: tuning forks, mouth organ, piano, accordion, tuning whistles, and the like. An audiometer can be used if it produces a continuous scale and not just the fixed octave frequencies. It is also easy to acquire the minimal knowledge of musical tones which is necessary for these comparisons of the patient's vocal qualities with the fixed tones of musical instruments. In any case, it is no more difficult than is the technique of auscultation and percussion which has to be learned by every medical student, although it requires refined auditory discrimination and considerable practice.

Another problem is the recording of the voice for objective demonstrations of therapeutic results. This is easily and inexpensively done with a magnetic tape recorder. Again, it is sufficient to use a fairly good instrument in a quiet room. High-fidelity systems or soundproof rooms may be ideal but are not necessary. One only has to bear in mind that the quality of any recording depends on the microphone, the actual recording system, and the reproducing speaker. Hence, one cannot rely on cheap models because their limited frequency ranges would suppress the higher overtones. These are important for the natural reproduction of pathologic voices because hoarseness is produced by abnormal laryngeal noises which contain many high overtones. Cheap recording systems, therefore, tend to subtract part of the hoarseness and make a hoarse voice sound better than it actually is, just as they subtract from the brilliance of a good voice and reproduce it poorly.

Scientific purposes naturally require more elaborate equipment. Pitch and frequency relations are demonstrated with the oscilloscope or its modifications, such as the tone-frequency spectrometer or visible speech recorder. Intensity measurements are made with the sound-level meter. Since the exhaled air stream is the driving force for all vocal performances, its registration and analysis furnish important information on all vocal functions. Luchsinger's methods of respiratory analysis determine the velocity and volume of the air consumed during a given vocal performance and establish its relationship with the measurable acoustic qualities of loudness, timbre, and vocal style. Strobos-

copy is important for diagnostic and theoretical purposes (registers, mode of vocal cord vibrations, etc.), while fluoroscopic and tomographic studies give further information on specific vocal problems in health and disease.

GENERAL QUALITY OF THE VOICE

A good criterion for the general quality of the voice is immediately available by determining the phonation time. This means the number of seconds during which a particular tone can be phonated on a given vowel with one exhalation. The patient is instructed to take a deep breath and to say "ah" (or "o," or "e") as long as he can. In general, phonation time varies between 20 and 30 seconds for vowels on tones within the median speaking range.

The physiologic and pathologic variations of phonation time depend on many factors: (1) respiratory factors: vital capacity, depth of inspiration, carbon dioxide tension of the blood; (2) neuromuscular factors: neurovegetative balance (Seemann), emotional state, physical activity; (3) laryngeal factors: gross vocal cord adduction, glottal closure during phonation, opening quotient depending on pitch and register (Doehne); (4) resonant factors: type of phonation (open, covered, free, throaty, etc.); (5) articulatory factors: open or closed vowels, nasal resonants; (6) general factors: age, sex, health, training, physical condition.

This interdependence of several factors can be seen in the mode of vocal cord vibration and the shape of the glottis, which in turn depend on pitch and register. To supplement the stroboscopic observations of various vocal performances of the normal human subject, W. Trendelenburg and coworkers studied the isolated cordal vibrations on the prepared cadaver larynx. The specimen was transilluminated from below and filmed from above, as the well-known illustrations of his "shadowgraph" procedure reveal. Besides the photographic tracing of the glottal openings and the electronic tracing of the cordal vibrations, he recorded the laryngeal sound.

First, it could be seen that the glottis remains closed for two-thirds of each period of low tones, whereas the closing time decreases with rising pitch. This leads to the definition of the opening quotient (O.Qu.) of the glottis as the time of each opening relative to the total time of each period. Hence, low tones show a small O.Qu., and high tones show an increasing quotient up to 1.0, when the glottis remains permanently open for falsetto tones. According to Tarnoczy (1951), there is a linear relationship between O.Qu. and frequency: 0.2 for 100 cps, 0.7 for 400 cps, etc.

It follows that phonation time is very short (a few seconds) when the cords cannot meet owing to laryngeal paralysis. Since most of the air escapes without being transformed into sound, each phonatory expiration is short, breathy, and ineffective. Hence, the patient is breathless and shows the well-known paralytic dysphonia. As he learns to improve the closing of his glottis, his phonation time increases again at the same rate as his voice improves. This simple test gives, therefore, a very impressive and objective demonstration of the vocal ability at any moment and its changes with time.

ACOUSTIC ANALYSIS OF THE VOICE

Since the voice is an audible phenomenon, it has to be described in acoustic terminology. This also clarifies the confusion of vocal nomenclature (Stern). The basic psychoacoustic qualities of the voice are correlated to physical properties, which in turn depend on the physiologic and anatomic characteristics of the individual voice organ. This correlation of (1) subjective audible impression, (2) objective acoustic structure, and (3) biophysical constitution is evident in all vocal phenomena. This paper gives only a rough outline of the basic vocal qualities, namely: pitch, loudness, timbre, and some of their subclassifications such as vocal range, registers, vocal styles, and musical voice types.

(*a*) Pitch depends largely on the frequency per second of the primary laryngeal tone. Low tones have a low frequency and

high tones are produced by many cycles per second (cps; in German Hz, after Hertz).

The frequency of vocal cord vibration is determined by the length, width, thickness, specific weight, and tension of the cords; by the shape of the glottal margins; by the subglottic pressure; in short, by the anatomic configuration and the physiologic action of the entire voice organ. For low tones the vocal cords are short and relaxed, while they are lengthened and stretched as the frequency increases. This is no contradiction to general instrumental principles, since the actual sound-producing mechanism, namely, the glottis, is long and triangular for low tones and becomes short and oval and moves forward with rising pitch. Slight fluctuations of pitch in the singing voice are called vibrato, while excessive oscillations of frequency and intensity are called tremolo (Sokolowsky, Sjöström).

The perfect function of the vocal cords is immediately disturbed by any disease or condition which changes their dimensions and shape. Temporary hyperemia due to hormonal, allergic, or functional causes easily disturbs the singers because it produces an increase in specific weight and thickness of the cords. The variable degrees of hoarseness during acute colds are the physical result of the well-known inflammatory changes (hyperemia, swelling, soreness).

(*b*) The vocal range reflects the ability to produce various laryngeal frequencies. In each person one distinguishes two ranges: the physiologic and the musical.

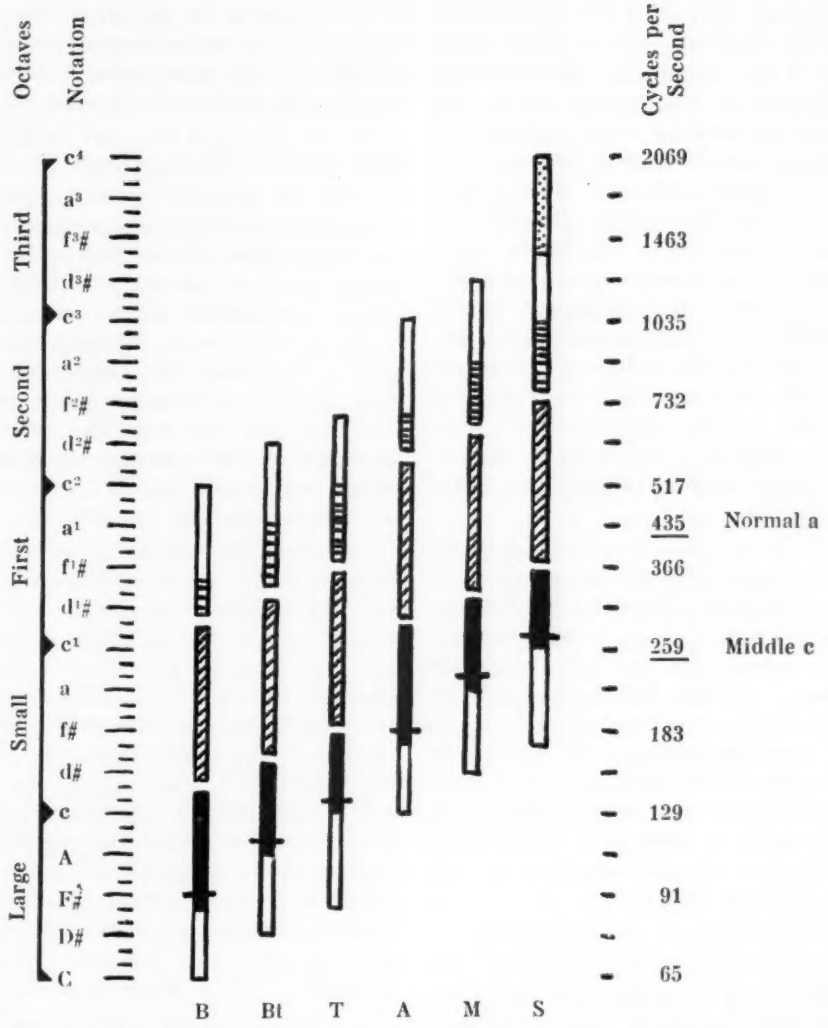
1. The physiologic range embraces all producible tones from the lowest and faintest chest tone to the highest falsetto or whistle tone under great effort; it covers two to four and a half octaves (Gutzmann, Nadoleczny, Preissler, Iida).

2. The musical range, which alone can be used artistically, is smaller and requires at least two octaves or two and a half to three octaves in very good singers. Certain publicity claims that some supposedly exceptional singers possess an unusual range of four to five octaves naturally have to be understood as meaning physiologic ranges, even though the lay public is misled to assume an unusual musical range. The vocal range is one of the paramount determinants of the voice type (Schilling). In low voices the range extends over the low tones and in the high voices over the high tones. The Figure illustrates these relationships in musical notation and acoustic cycles.

3. It also shows a third concept of range, namely, the human range, if one combines all individual ranges from the lowest bass to the highest coloratura soprano. This collective range of all human voices spreads over five to five and a half octaves, or from about 50 to 2000 cps (G_I-c^4).

(*c*) The median speaking range lies at the lower end of the musical range. It comprises five or six tones. Thus, a baritone voice fluctuates between F (80 cps) and c (129 cps) with a center around A (110 cps) for normal conversation. All voice types have their own characteristic ranges of the spoken voice tones. Emotional and environmental influences, such as convention, fashion, or personal preference, easily modify the natural conditions. American women at present prefer rather low speaking ranges, namely, those of the female contralto voice, and sometimes even descend to the natural speech range of the high male tenor. Male radio announcers cultivate an abnormally low and "booming" voice which descends as low as C (65 cps). Average physiologic values are shown in the Figure.

(*d*) Vocal registers are an undeniable phenomenon of every normal voice. It has been postulated that the good singing voices have no audible registers and that their noticeable presence means a faulty vocal technique. This definition of the musical "one-register theory" does not refer to the normal untrained voice; rather, it explains the goal of every vocal training: the equalization of registers and the elimination of breaks at the transitions, so that all tones can be sung smoothly without any sudden interruption of tonal continuity. Even then, the



Relationship of registers, range, average speaking pitch, and extreme tones of the human voice: B indicates bass; Bt, baritone; T, tenor; A, alto; M, mezzosoprano; S, soprano. Black segments of columns show chest register; diagonally lined segments, middle register, and horizontally lined segments, head register; all three segments indicate musical range. Lowest and highest blank segments show physiologic range. Punctuated portion above soprano range indicates whistle register. Segments with horizontal lines are average speaking ranges. Octaves and notation according to von Helmholtz. Frequencies based on the old concert a (435 cps); the present standard a is 440 cps.

trained ear can detect fine differences in tonal quality of low, medium, and high tones (Bellussi, Luchsinger).

The physiologic definition was first given by Manuel Garcia, who distinguished three registers. Nadoleczny augmented this definition: "Registers are a series of consecutive voice tones of equal timbre; the musical ear can distinguish them in certain tone regions from another adjoining series of tones; this acoustic resemblance is due to a constant distribution of partial tones; registers are the audible result of discrete vocal mechanisms. . . ." Every voice shows three main registers: chest, middle, and head register.

In addition, there are a few subdivisions: the church bass register below the chest register of very deep bassos; the male head voice appears in three forms, namely, the loud full tone of head voice (Luchsinger), the delicate artistic falsetto, and the thin untrained falsetto of the natural voice; and in the female there is the flageolet (or whistle) register above the highest head tones of the coloratura soprano (Lüders, Garde, Schultz). The knowledge of the physiologic (untrained) falsetto voice is important for practical purposes because the falsetto voice may appear in many pathologic conditions: habitual (postmutational), psychogenic, traumatic (shortening of glottis), paralytic, and cerebral (diencephalic, extrapyramidal, etc.) states.

Much information is already available on the details of laryngoscopic (Katzenstein), stroboscopic (Stern, Tarneaud, Musehold, Panconcelli-Calzia), acoustic, pneumographic, or radiologic analysis of the register differences (Pielke, Sokolowsky, Scheier, Nadoleczny, Luchsinger, and others). To avoid misunderstandings, it should be stressed that the registers are always gradually mixed in a well-trained voice. Some authors prefer to speak of a "predominant chest register" or "predominant head register" (Winckel, 1952).

(*e*) Vocal intensity is correlated to the amplitude of the vocal cord vibrations. This depends partly on frequency: Low tones have a wider amplitude than do the high tones. The most important determinant of vocal sound pressure is the subglottic air pressure.

For the objective measurement of vocal intensity, Luchsinger had described an electroacoustic method. While observing with a stroboscope the vocal cord amplitude through a telescopic measuring lens, he registered the voltage of sound pressure. Weak tones showed an amplitude of 0.5 to 1 mm. and a sound pressure of 1 to 2 volts. Loud tones increased the amplitude to 3 mm. and the sound pressure to 9 to 10 volts. Within limits, this reflects the proportion of intensity to the square of the amplitude. Similarly, the specific air volume increases during a gradual crescendo in proportion to the square root of sound pressure (Vogelsanger).

(*f*) The timbre of the voice corresponds to the spectrum of partial tones. Their suppression by damping or enforcement by resonance occurs in the supralaryngeal cavities. Since these cavities show great individual variations on the one hand, and familial similarities on the other, their configuration modulates the individual character of the voice. In addition, they are under the constant influence of the cortical, emotional, and neurovegetative centers of the brain, so that emotional states readily become manifest in typical shades of the voice: anger, joy, fear, security, etc.

Luchsinger demonstrated a gradual series of vocal styles and their interrelationship with corresponding changes in the vocal organs. On the one side there is the guttural, suppressed, and faint sound of ventriloquism, which is produced with maximally constricted and narrowed resonance chambers (Pressman's sphincter action). The next step is open singing (bel canto style), which shows a considerable widening of the resonators. Covered singing (German dramatic style) brings about a further enlargement of the throat cavity in all directions. The other extreme is represented by yodeling, which requires a maximal widening of all supralaryngeal resonators.

(*g*) Voice types are as well known as the various body types. For centuries one has distinguished bass, baritone, and tenor in the male and contralto, mezzosoprano, and soprano in the female.

The individual voice type is as much a part of the general psychophysical constitution as is any other human quality. For this reason, the opera art has developed standard types for its dramatic parts, which are sung by particular singers who possess the appropriate voice type, vocal technique, physical appearance, and artistic inclination. Accordingly, each of the six voice types is subdivided into three groups of dramatic,

lyric, and buffo (or coloratura) roles or parts. The correlation between constitution and voice type was studied by Schilling, who confirmed the empirical impressions.

REPORTING OF PHONIATRIC FINDINGS

With the above information in mind, one can proceed to the actual examination of a patient. It may be done in a short and simple manner for daily practice or with elaborate refinement for medicolegal purposes, as reported in a previous paper (1951).

After laryngologic examination has clarified the underlying condition, one observes the general mode of phonation: regular, hyperkinetic (with excessive movements of adduction), hypokinetic (incomplete movements), spastic, uncoordinated, etc. A discrepancy between the sound of the voice during reflex phonation (coughing, gagging, clearing of the throat) and during voluntary phonation suggests a psychogenic disturbance.

The general quality of the voice is described as clear, husky, or hoarse; loud or weak; full or breathy; pleasant, shrill, muffled, etc., and in any other terms that might best illustrate any particular quality.

Phonation time is recorded next. After a few trials, the patient takes a deep breath and phonates a vowel on a comfortable pitch level as long as possible. While a normal young man may hold the vowel "o" on a comfortable tone of his speaking range, for instance, the tone A=110 cps, for 25 seconds, a patient with laryngeal paralysis may produce the vowel "o" on tone a=220 cps (too high) for only 5 seconds. This is noted: O/a=5 sec.

While the patient tells his story, the average speaking range of his conversational voice is compared with a tuning fork, piano, etc. One may notice again that this patient speaks on a high pitch, almost with a falsetto voice. This fact is then recorded: conv. around a (220 cps).

Next one tests the expansion of the total voice range. By letting the patient hum a downward scale, one finds the lowest limit of his voice. Similarly, he hums or sings an upward scale until the highest tone is found. If the voice is fairly good, one can hear the register transitions. In a poor voice they are, of course, obscured by the general hoarseness. Whereas a normal baritone voice may show the range and register formula of A-e, e-e^1, e^1-a^1, this imaginary patient may have the following range limits: d-c^1, no transitions.

Finally, the higher vocal productions, such as the ability to sing, recite, or call aloud, or any professional use of the voice, are tested. These may be limited or totally absent.

SUMMARY

After stressing the importance of a functional assessment of the voice for objective comparisons of functional results of laryngeal surgery, a brief outline of voice physiology is presented. Instrumentarium and procedure of a brief method of vocal analysis are discussed. The findings of such phoniatric examinations are recorded in a simple but objective formulation which is based on musical usage and terminology. While a minimal interest in, and knowledge of, musical notation is necessary, this experience can be acquired after very short practice.

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News and Comment

GENERAL NEWS

Specialists Certified by the American Board of Otolaryngology.—The following physicians were certified as specialists by the American Board of Otolaryngology at the recent examination held in Chicago, Oct. 3-7, 1955:

Bautista, Ariston G., Brooklyn 3.
Beatrous, Frank Theo., New Orleans 12.
Bennett, Truett Vann, Atlanta.
Berard, William P., U. S. N. H. Navy 926, % F. P. O., San Francisco.
Brown, Aubrey K., Jr., Washington, D. C.
Burns, Stanley S., Jr., Charlotte, N. C.
Campana, Joseph F., Williamsport, Pa.
Chiang, Tung Ming, San Francisco.
Cope, Beverly A., Ashland, Ore.
Davis, Albert J., Jr., Louisville 16.
Douglas, John E., Austin 1, Texas.
Dragovich, Jerry J., Bremerton, Wash.
Draper, William Leonard, Houston, Texas.
Eberly, Byron T., Portsmouth, Va.
Ewell, Murlin Knox, Bryn Mawr, Pa.
Fragola, Anthony F., Middle Village 79, N. Y.
Gafford, August V., Rome, Ga.
Glaeser, John H., Minneapolis.
Green, Oscar, Indianapolis 8.
Harrison, Wiley H., Chicago 2.
Hendrix, William C., Jr., Fort Worth 4, Texas.
Howell, Julius Ammons, Winston-Salem, N. C.
Knight, Frederick Edward, Fresno, Calif.
Kusske, Bradley W., St. Paul.
Lake, James Douglas, Bay Shore, N. Y.
Lee, Samuel, Los Angeles 7.
Lerner, George G., Syracuse 2, N. Y.
Lewis, Robert C., Sacramento, Calif.
Malinoski, Mitchell V., Poplar Bluff, Mo.
McIntire, Frederic Joseph, Jr., Lynn, Mass.
Moran, John Patrick, Corpus Christi, Texas.
Morgan, William C., Jr., Charleston, W. Va.
Mulcahy, Neil Donald, San Francisco.
Myers, Paul B., Portland 5, Ore.
Paine, Albert J., Bluefield, W. Va.
Post, Edmund A., St. Paul.
Quilty, Thomas James, Goshen, Ind.
Ralston, Robert L., Spartanburg, S. C.
Reagan, Daniel Joseph, West Boylston, Mass.
Reese, Stanley E., La Mesa, Calif.
Shuster, Allen R., Philadelphia 3.
Smith, Denton Weymouth, Beaumont, Texas.
Songco, Vincente F., Brooklyn.
Trible, William M., Washington 6, D. C.
Twichell, Gilbert A., Iowa City.
Walter, Claus D., Columbus, Ohio.
Weiser, Albert, Fall River, Mass.
Yast, Charles J., Gary, Ind.

Acute Poliomyelitis of the Mother, with Aural Lesions in the Premature Infant

GEORGE KELEMEN, M.D., Boston

Pregnancy is recognized as one of the factors that promote the development of poliomyelitis. Anderson (1948) quoted statistics according to which the incidence of polio in pregnant women was twice or three times the expectancy. Polio in the last trimester was more serious than in the first six months. Aycock (1946) related that pregnancy predisposes to acute polio, with a tendency for the disease to occur in the first trimester in women carrying a male and in the third trimester in those carrying a female fetus.

Evidence to the effect that the polio virus cannot break through the placental barrier was advanced from several sides (Huerny, 1942; Froevig, 1947; Stokes, 1948; Blechmann and Oliveau, 1948). It would seem that not much danger awaits the progeny from direct transmission of the virus during gestation. But Aycock (1941) suggested that infection can be initiated during pregnancy, as paralytic polio was observed in infants from the fifth day on (Froevig: fourth day). Considering the incubation period for polio, it is hard to see how infants showing signs of the disease within two weeks after delivery could have acquired the virus outside the uterus.

Regarding the influence of polio on the ear, very few data have been forwarded so far. Paul (1951) reported that curious jerky ocular movements, "nystagmoid rather than a true nystagmus," are described with increasing frequency in the polio attack. Zausmer (1951) recorded frequent paralysis of the facial nerve in polio. In both cases central as well as peripheral origin may be assumed. Lederer (1952) gave points of differential diagnosis between lateral sinus thrombosis and polio. Bollobás (1955) discovered eight times—not further specified—ear complications among 125 autopsied polio cases; 2 were unilateral and 6 bilateral; in their frequency they equaled the pulmonary (8) and surpassed the cardiovascular (6), the enteric (2), and the septic (2) complications. All in all and contrary to the momentous significance, especially of the bulbar form, in causing pharyngeal and laryngeal symptoms, the hearing organ seems to stand outside the general picture of the disease.

In view of the devastations by rubella in early pregnancy, Anderson, Anderson, Skaar, and Sandler (1952) followed 53 children born of mothers with polio, by nursing visit or by correspondence. Only two reported a congenital defect, and this in no connection with the ear.

On the other hand, Aycock (1941) reported three premature deliveries in polio, all with death of the child (one in the sixth month, two without indicating the stage of pregnancy). Prematurity was and is considered, next to intracranial, spinal, or visceral trauma, and anoxia, one of the main causes of perinatal mortality (Potter, 1954). In its damaging influence on the hearing organ, prematurity was thoroughly analyzed by Voss (1923). Although premature delivery of mothers in or after an attack of polio is said not to show a higher incidence than its occurrence in women without polio,

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the peculiarities of this disease might well transform it into a complication of formidable pathological significance. Pregnancy complicated with polio is a common occurrence; premature deliveries were observed in a number of cases, and many of these resulted in death. Whether the improvement of the survival rate for premature infants, as registered for the last few years, extends to those born by mothers with polio remains to be taken up in detail.

Gruenwald (1949) stressed the role of virus disease of the mother in causing injury to the progeny and remarked that only a very small number of these cases have been examined at autopsy.

The data on the hearing organ of a premature infant born to a mother in an attack of polio are given here and represent, to my best knowledge, the first report of its kind.

REPORT OF A CASE

The 32-year-old white female patient was admitted to the Haynes Memorial Hospital on Oct. 31, 1953. Her first child was stillborn (placenta previa); after this she had four children, all living. She was in good health and in the sixth month of her pregnancy when she developed, six days before admission, shaking chills. Four days after admission severe pains started in the posterior aspect of both thighs.

Physical examination by Dr. Louis Weinstein, staff physician, revealed normal drums with physiological landmarks. Tonsilloadenoidectomy had been performed at 5 years of age. There was weakness of the hip, limbs, ankles, and toes on both sides and nuchal rigidity. She was Rh-negative, her husband Rh-positive.

On Dec. 10 she was delivered (expected day of confinement: Feb. 20, 1954) after 12 hours of labor pain of a boy weighing 2 lb. 13 oz., in right occiput anterior presentation, with the cord about the neck. No forceps and no anesthesia were applied. The newborn required artificial respiration, suction, and oxygen for maintenance, but breathed after 10 minutes without aid. He was placed in an incubator and transported to the Children's Medical Center, receiving oxygen on the trip.

The mother was discharged on Jan. 31, 1954, with the final diagnosis: spinal paralytic poliomyelitis involving the lower limbs, especially the right.

The infant died 44 hours later. The postmortem examination found marked prematurity, with a length of 38.5 cm., compared with a normal of 49 cm.

The pertinent part of the autopsy report (Dr. Sidney Farber) follows: The ears were normally formed and the external auditory canals were patent; the nasal passages were clear. The entire base of the brain was covered with a thick layer of clotted blood, which was subarachnoid in location and measured up to 3 to 4 mm. in thickness. The ventricular system of the brain was completely filled with blood. A rupture was found in the vena terminalis, and direct continuation established from the bleeding point in this vein led into the ventricular blood. Histologically, besides the fresh subarachnoid hemorrhage, congestion was observed in the vessels of the meninges and of the cerebellum.

No evidence of polio was found microscopically in the neuromuscular system.

The eye pathology report (Dr. Parker Heath) noted extensive hemorrhage in the orbit and the periorbital fat, engorged choroid, mild vitreous hemorrhage, and extensive retinal hemorrhage.

The final diagnosis was massive interventricular and subarachnoidal hemorrhage; chronic aspiration pneumonia; partial involution of the thymus; severe pulmonary hemorrhage and congestion; hypoplasia of all organs; immaturity of the kidneys, intestine, brain, and lungs.

Dr. Farber's discussion of the record explained that all three—the intracranial hemorrhage, the pulmonary hemorrhage, and the chronic aspiration—plus the extreme prematurity of the infant could adequately account for death. "The lack of evidence of poliomyelitis is not remarkable. In most of the cases of neonatal polio that have been reported the mother has been in the acute phase of her disease or in early convalescence at the time of the birth. It cannot be stated whether the infection is carried through the placenta or the infection is acquired during the passage through the birth canal. If the incubation period of polio in the newborn were known it could be better stated how the infection might be transmitted."

Through the courtesy of Dr. Farber, the right temporal bone, fixed in formalin, was received to undergo histological investigation. Decalcification, embedding in pyroxylin, and serial sectioning in a parahorizontal plane were followed by staining with hematoxylin-eosin and Heidenhain-Mallory and impregnation after Bodian. Microscopy included polarization and phase contrast. (The histological preparation was done by Miss Dorothy Linden; the microphotography, by Mr. Richard St. Clair.)

The dura was normal, without engorgement or thickening, with exception of the points enumerated below.

The tegmen and the petrous tip were diploic in character.

The middle ear (Fig. 1) was opened at removal from the cadaver and the tympanic membrane was lacking. The epitympanum was still filled with

Fig. 1.—Middle ear: Large ossicles (upper right) still embedded in mesenchyma. Blood enters from tubal corner (left). In mesotympanum (center) crura of the stapes and fibrinous mass. Heidenhain-Mallory; reduced 2/5 from mag. × 9.

mesenchyma, showing normal vascularization; while its mass surrounded the incus, the malleus was free to a greater extent and so was the medial aspect of the incudomalleolar articulation. The malleus enclosed, under a thin and continuous corticalis, a single large central marrow space; in the incus, several osseous partitions intersected the corresponding marrow-filled area. A conspicuous cartilage nucleus was seen located under the incudal surface of the articulation.

Near the tegmen, in the vicinity of the area of the subarcuate fossa, a cartilage block was persisting, in a stage of intensive ossification by penetrating osteogenic vessel buds.

The stapes showed very wide marrow spaces; a large marrow space was located in the cochlear capsule opposite the stapes plate. With the annular ligament intact, the niche of the oval window was filled with an organizing cushion divided into large cystic spaces. Engorged mucous membrane with small, scattered extravasates covered the crura and the head of the stapes. The niche of the round window (Fig. 2) was lined with a hyperemic mucous membrane, its lumen occupied by a hemorrhagic transudate.

Solid blood penetrated from the tubal orifice and filled entirely the tubal corner of the middle ear (Fig. 1). Bloody suffusion invaded the tissue of the tensor tympani muscle in its semicanal. Between the massive bleeding entering from the tube and the mesenchymal filling of the epitympanum, the center of the tympanic cavity was occupied by a dense fibrous mass.

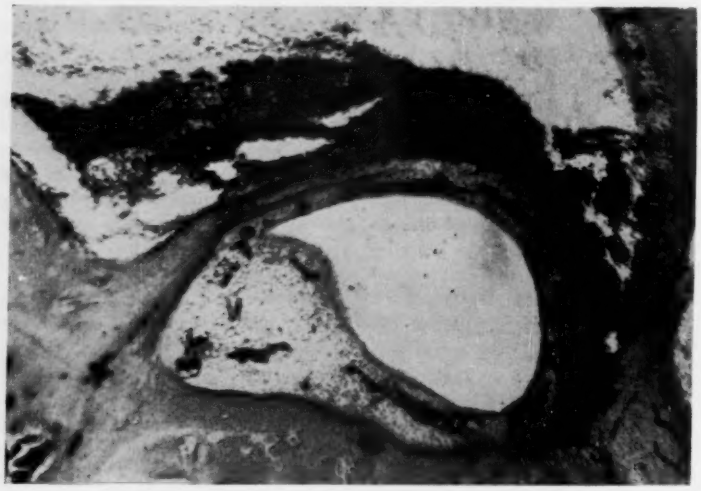

Fig. 2.—Blood enters through the cochlear aqueduct (left), spilling over the cochlear surface of the membrane of the round window. Engorgement in the thickened mucous membrane lining of the niche of the round window. Heidenhain-Mallory; reduced 2/5 from mag. × 50.

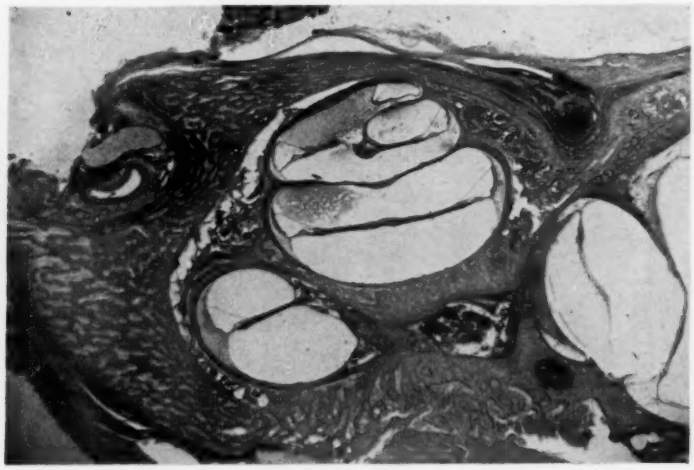

Fig. 3.—Cochlea with serofibrinous transudate in the middle and the apical turn. Sanguineous infiltration of the dura (upper right corner) in the facial hiatus, and of the facial nerve. Heidenhain-Mallory; reduced 2/5 from mag. × 9.

In the inner ear, the otic capsule was developed in concordance with the seventh gestational month. The periosteal and the endosteal layers of the cochlear capsule were continuous, while the endochondral layer still contained wide marrow spaces (Fig. 3).

In the vestibular area, fibrin filled the utriculus; the sacculus, somewhat dilated, was free. The superior semicircular canal contained fibrinous transudate with infrequent extravasates in the endolymphatic and perilymphatic spaces; the nerve epithelium and the cilia of its ampullar crista were intact (Fig. 7), while a sequestrum of the cupula, corresponding to its distal half, was seen lying free in the lumen of the ampulla amid serofibrinous transudate, with red blood cells clinging to the free edges of the sequestrum. Fibrin filled the endolymphatic and perilymphatic spaces of the posterior semicircular canal together with scattered small extravasates, while the canalis singularis contained free blood. The endolymphatic sac was collapsed.

In the cochlea, the vestibular and, to a lesser extent, the tympanic scalae of the middle and upper turns contained fibrin (Figs. 3 and 4). The cochlear duct was free except for some serofibrinous transudate over Corti's papilla laterally from the tunnel. The membrane of Reissner (Figs. 3, 4, and 5) preserved everywhere its normal position. The tectorial membrane was hovering above the papilla and was clearly touched by the cilia of the hair cells (Figs. 5 and 6). The papilla of Corti was everywhere intact except that at some points it seemed to be slightly infiltrated by transudate without change as to its configuration (Figs. 5 and 6). The vessels of the stria were filled to a normal degree. Through the gaping cochlear aqueduct

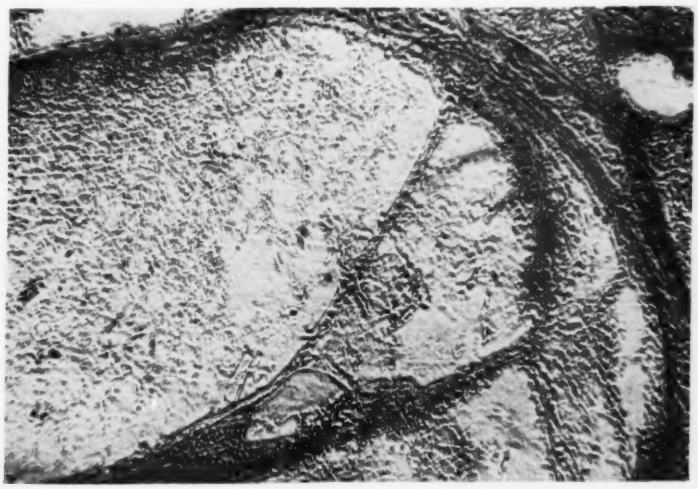

Fig. 4.—Cochlea with serofibrinous transudate; phase contrast; reduced 2/5 from mag. × 100.

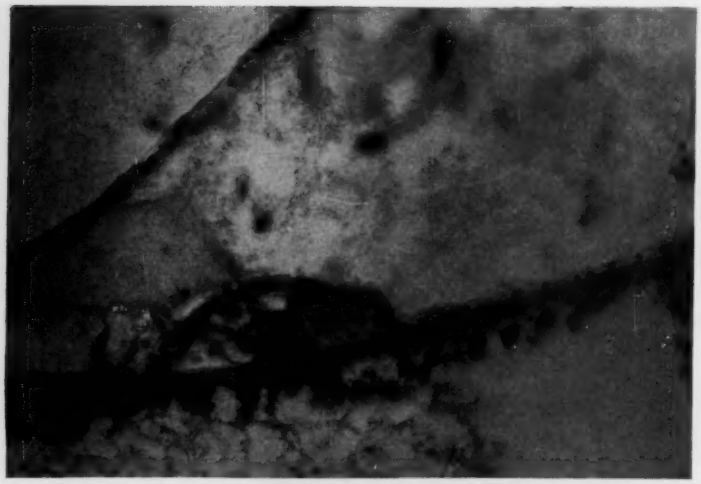

Fig. 5.—Cochlear duct with partial slight serous infiltration. Fibrin in the tympanic scala. Hairs of external hair cells in contact with the tectorial membrane. Hematoxylin-eosin; reduced 2/5 from mag. × 200.

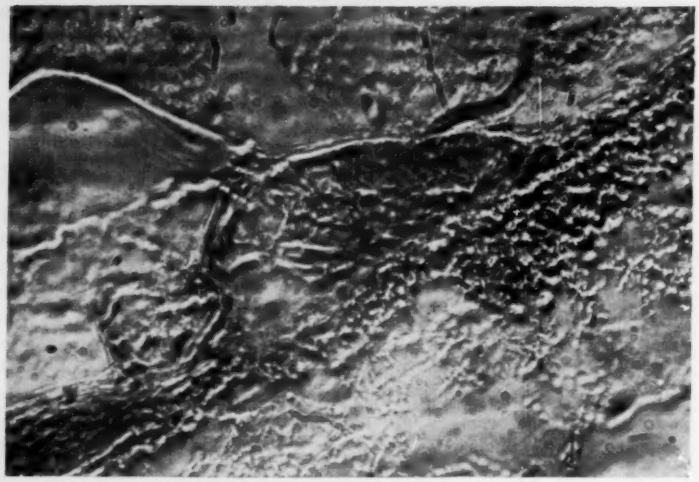

Fig. 6.—Section comparable to that of Figure 5. Hairs of external hair cells in contact with tectorial membrane. Phase contrast; reduced 2/5 from mag. × 400.

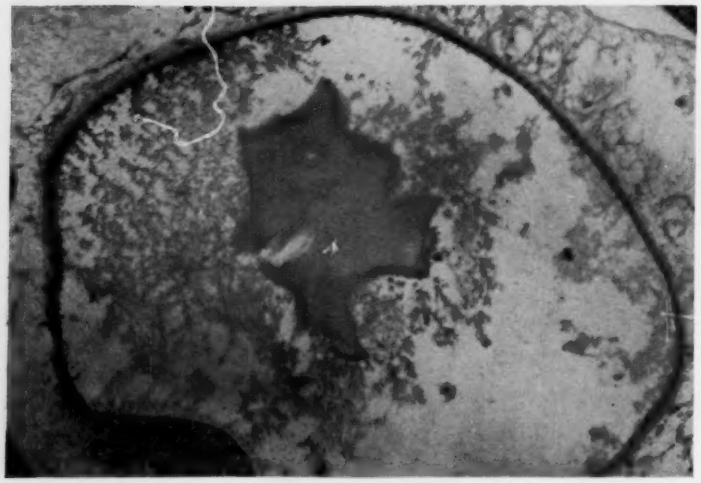

Fig. 7.—Ampulla of the superior semicircular canal. Crista, including hairs intact, and cupula sequestrated by the serofibrinous transudate. Heidenhain-Mallory; reduced 2/5 from mag. × 50.

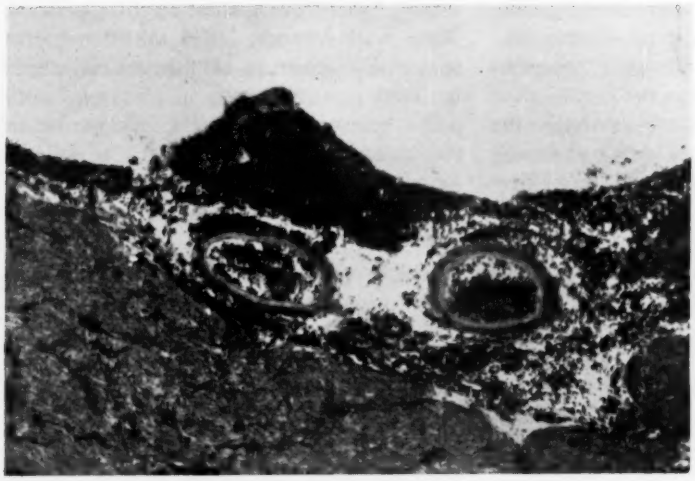

Fig. 8.—Inner acoustic meatus. Engorgement in the vessels of the nerve sheath. Extraneural and intraneural extravasation. Heidenhain-Mallory; reduced 2/5 from mag. × 100.

blood entered the tympanic scala and spilled over the tympanic surface of the membrane of the round window (Fig. 2). The modiolar vessels were engorged.

The fundus of the inner acoustic meatus contained blood, with the dural lining of the meatus marked by engorgement and extravasates. Hemorrhage was present perineurally as well as within the trunk of the eighth nerve (Fig. 8).

The veins of the pericarotic plexus were engorged with some extravasation.

The dura around the hiatus of the facial canal was suffused with blood, with hemorrhage present in the facial nerve and in the geniculate ganglion.

Summarizing the findings in the temporal bone: The developmental stage corresponded to the seventh gestational month, with appropriate formation of the layers of the cochlear capsule and with mesenchymatous cushions still filling wide areas of the epitympanum. In the presence of a serofibrinous otitis media and interna, the end-organs of the inner ear remained intact, with the exception of the sequestrated cupula of the superior ampullar crista. The outstanding feature was the presence of massive blood, penetrating from (*a*) the tube, (*b*) the hiatus of the facial canal, (*c*) the cochlear aqueduct, and (*d*) the inner acoustic meatus. A collateral engorgement, with multiple extravasates in the mucous membrane lining of the tympanic cavity and of the inner acoustic meatus, and bloody suffusion of the body of the tensor tympani muscle completed the picture.

COMMENT

Hellman (1952) remarked that, in the present day, talking of birth injuries does not mean, as it did 20 years ago, merely the result of a direct trauma to the baby: The base has to be broadened to include late pregnancy hemorrhages, anoxia, and dystocia.

The oldest reference to a congenital hemorrhage into the labyrinth is Toynbee's (oral communication to Moos, 1866). A number of sporadic communications followed until the comprehensive report of Voss (1923) gave a new impetus to further investigation. In view of the here-presented case it is of relevance that among his series of 12 histologically examined petrous bone hemorrhages not less than 7 originated in the prematurely born. Among the 80 pairs of temporal bones enumerated by Albrecht (1930) which underwent histological examination after aural birth trauma 27 were found in the prematurely born.

The most important of the conclusions of Voss was perhaps the warning against making any sharp distinction between antepartum and intrapartum origins of hemorrhages, as both appear clinically to be equally "congenital." Furthermore, Gruenwald (1949) declared all classifications recognizing two distinct categories (malformation and disease) as arbitrary, as there is no inherent difference between the two; autopsies in stillborn and newborn babies revealed an unexpected high incidence of typical disease processes in the embryo. Potter (1954), combining the periods during the delivery

act and the first days after delivery, discussed them under the term "perinatal." She related that the pathologic processes that may affect the infant in the first 10 days after birth are few and are especially the result of anoxia or trauma sustained during passage through the birth canal, or of premature delivery.

While stating that intracranial hemorrhages might be present at delivery in the second trimester, Potter and Adair (1940) found, among the causes of death at delivery in the last trimester, intracranial hemorrhages in 17.4% and prematurity without demonstrable pathological changes in 8.6%. They declared fractures of the base of the skull to be extremely infrequent. Similarly, Ingraham and Matson (1954) found fractures of the cranial base in infancy and childhood as being "not common." However, it has been shown (Kelemen, 1944) that without serial sectioning of the temporal bones fractures of this kind can easily go undetected, and that practically without exception they extend to the surrounding bones of the cranial base.

By applying suction in the area of the longitudinal sinus, Berberich (1925) experimentally produced hemorrhages in the temporal bone. He established the progress of the stasis as follows: longitudinal sinus, transverse sinus, superior and inferior petrous sinuses, venes of the temporal bone.

Hemorrhages occurring in late pregnancy were discussed according to their different types by Hertig (1952).

Vessel damage can be based on changes in the walls of capillaries and their increased permeability with extravasation and edema.

A lower resistance of the vessels was already suspected by Albrecht when he saw bilaterality of the changes in 70 of the 80 pairs of temporal bones tabulated by him. Vascular lesions as basis of damage to the ear are to be recognized without doubt in syphilis (Kelemen, 1929) and can be postulated in diabetes (Kelemen, 1955) but are of less help in explaining damage to the progeny in polio, except in a mechanical sense. Apart from qualitative changes in the vessel wall Albrecht called attention to the anatomic disposition of the vessels within the inner acoustic meatus as a basis for accidents, because of insufficient development or incomplete anchoring in an early period; he found bleeding always at points of the entrance or exit of vessels into or from the bone.

The dangers inherent in prematurity have been recognized by many. According to Hellman, it is generally accepted as the largest single factor contributing to infant mortality, about half of the fatalities being due to this cause. Voss explained that premature birth is easy on the mother but perilous to the child; the pressure change occurs with catastrophic rapidity and with intracranial hemorrhage in its wake; it is a serious insult, with the auspices for survival decreasing the younger the newborn. Phelps (1950) observed that in the premature infant the blood vessels in the brain are more readily broken than they are in full-term infants, and surmised that this rupture may result, during a precipitate delivery, from a too rapid drop from an extremely high intrauterine pressure to atmospheric pressure, as well as from actual trauma. Prematurity can be accompanied by conditions as anoxia, kernicterus, toxemia, and others, each of which, according to Fish (1955), singly could be a cause of deafness.

Turning to the factor of dystocia the period between the attack and the pregnancy deserves attention. Arriving one year or more after the attack, a normal offspring can be anticipated (Kleinberg and Horwitz, 1941). But it must be added that pelvic deformities after polio will be of more and more consequence in later years, with resulting complications at birth.

Besides the influence of excessive head molding it might be well worth while to study the mode of presentation in its possible relation to ear injury. The two sides of the head are exposed to different strains within the birth canal. This factor, next to asymmetric application of forceps, can account for unilateral functional damage.

Farkas (1952) suggested among the causes for the rapid increase of polio—through sensitization of the organism by mobilization of the strain—the too generous use of the forceps besides surgery in general, and the effect of anesthesia. Although forceps or anesthesia were not applied in the present case, as a general consideration damage to the ear by both factors is well within the bounds of possibility, in case of the first by direct injury, of the second through changes in oxygenation.

The findings as described in this presentation conform widely to the pathological picture of mechanical birth trauma to the ear as established mainly by the work of Voss and Albrecht. Similar penetration of massive blood through the cochlear aqueduct was observed by Grünberg (1922) in a case of widespread subarachnoidal hemorrhage.

Concerning prognosis, the lesions have to be classified into a reversible and an irreversible group. The engorged vessels of the mucous membrane in the middle ear would not have caused permanent damage, and the small extravasations for which they served as a source would have been quickly reabsorbed, possibly even without leaving a thickened, fibrotic membrane. But it must be borne in mind that a similar early damage to the mucous membrane of the tympanic cavity was considered to be a predisposing factor for the development of an otitis media in the newborn, while opinions regarding the occurrence of an intrauterine serous meningitis—as shown by engorgement in the dura in circumscribed areas—are still in disagreement. A sign pointing to a possible future functional loss is to be seen in the beginning organization, with bands and cystic spaces filling the niche of the round window. Healing, with fibrosis of the injured tensor tympani muscle, could have influenced the mobility of the ossicular chain.

The massive hemorrhage entering from the Eustachian tube would have been reabsorbed or expelled by the penetrating air.

The light serofibrinous accumulations in the middle and the inner ear would have quickly disappeared, without further consequences. Wittmaack (1926) declared the serous-fibrinous labyrinthitis to be, in strict pathological sense, a process restricted to the perilymphatic space. This can be considered one more reason to exclude functional damage for this source.

On the other hand, permanent damage can be predicted by the presence of hemorrhages in the inner acoustic meatus, with destruction within the eighth nerve. Functional loss might have followed equally the conditions found within the facial nerve. Damage to the latter as a frequent finding in polio was mentioned above.

SUMMARY

The right hearing organ of a 7-month premature infant, born 72 days prior to expectancy to a multipara in the 40th day of her attack of paralytic poliomyelitis, is described on the basis of a histological sectional series. The child died 44 hours after delivery, and autopsy revealed massive subarachnoid hemorrhage, pulmonary hemorrhage, and bronchopneumonia.

The elements of the temporal bone presented the adequate developmental stage. Massive bleeding was seen entering from the tube (originating from the pulmonary hemorrhage), the hiatus of the facial canal, the cochlear aqueduct, and the inner acoustic meatus (in the last three locations as continuations of the extensive subarachnoid hemorrhage). Extravasates caused destruction in the acoustic and in the facial nerve.

The hemorrhagic manifestations point to a damage to the vessels by the strain of the perinatal occurrences, with a rupture clearly demonstrated in the vena terminalis.

Pregnancy is recognized as one of the predisposing factors for poliomyelitis. Birth trauma to the hearing organ is to be considered in relation to well-documented deleterious consequences of prematurity to the ear, and later by the higher incidence of injuries from pelvic deformities in the wake of polio attacks.

Anatomical damage, as in the present case, and danger from other sources like anoxia suggest that more attention than that given

hitherto should be paid to possible functional lesions in children delivered in acute polio of the mother or amid complications from pelvic deformities caused by an earlier attack of the disease.

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Further Psychosomatic Aspects of Otolaryngology

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For years I have observed all kinds and categories of ear, nose, and throat complaints, and in my opinion most of them were real. To detect organic pathology in the field of otolaryngology is a fairly simple task; to prorate accurately the importance of organic and functional disease coexisting in the same patient is more difficult; but to state with assurance that all a patient's complaints are functional in nature is to assume a position based on circumstantial evidence that is easy to believe (and may be correct) but is most impossible to prove. Psychosomatic disorders in the field of otolaryngology run rather true to form. Only in exceptional instances where circumstances causing a psychosomatic disability are unusual do we have embellishments that make a particular patient of unusual interest.

The run-of-the-mill complaints of psychosomatic disease that come regularly into the office of an otolaryngologist we classify as follows: (*a*) common forms of psychosomatic manifestations and (*b*) uncommon forms of psychosomatic manifestations.

Although the origin of either form may be a phobia, the cause is often recognized or unrecognized "psychosomatic repression."

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A. The common forms of psychosomatic manifestations, some of which we see almost every day, are observed in patients with (1) a lump in the throat; (2) a tightness in the neck; (3) a burning sensation about the mouth and pharynx; (4) a loss of voice; (5) a difficulty in swallowing, and (6) a dizziness or lack of balance.

B. The uncommon forms of psychosomatic manifestations are worthy of particular consideration, not because the mechanism producing them is different from the common form, but because the psychological background is more challenging—that is, from the point of view of diagnosis and treatment.

CASE 1.—A boy and his father came into the office because the son had a stuffy, running nose. I suspected a psychic rhinitis and referred him to a neuropsychiatrist. The boy worked after high-school hours in a tobacconist's shop. Each week for several months he had stolen from the employer's till $3 to $5 in cash and when I saw him he was still doing it. The lad felt compelled to continue this embezzlement, because he had established among his boy and girl friends the reputation of being a good spender; and he felt compelled to discontinue the embezzlement, because he knew that as soon as the auditor examined the books, he would be caught and "probably be sent to the reform school."

When the cause of the rhinitis was explained to him by the psychiatrist, the nose symptoms disappeared, but the last we heard he was continuing his embezzlement practice and had psychosomatic symptoms located in other parts of his body.

CASE 2.—For more than a year a woman 65 years of age had complained of "a terrible pain in her throat." In spite of negative physical findings, negative x-ray examinations, negative laboratory studies, and innumerable consultations, she presented no organic cause for her distress. Although I have observed her several times, I have never seen her show any tangible evidence of pain, but she insisted that it was always with her. Strong anodynes gave her no relief.

One day, quite accidentally, she gave me the answer to her complaint. A year previously she had undergone a cataract operation with a "perfect result." The ophthalmologist advised an operation on the other eye. Unguardedly she asked me this question, "Don't you think it would be dangerous for me to have an operation on the other eye so long as I have this terrible pain in my throat?"

In other words, she was using the pain in the throat as an excuse to keep from having the other cataract removed.

CASE 3.—For almost 18 months I had under observation a school girl, 11 years old, whom her teacher had suspected of having a loss of hearing. The teacher's suspicion was borne out by school audiometric tests, but when the child was booked for attendance in a school having conservation-of-hearing classes, the father objected, saying that the child was not deaf and that the department of special education was in error about the whole matter, that the girl heard all right at home and at church and everywhere except in school. My acoustic examinations were too inconsistent to make a diagnosis of organic deafness. After she was removed to a parochial school, however, repeated acoustic tests (over a period of four years) have consistently shown normal hearing. This four-year observation period with no evidence of deafness has apparently established the correctness of the father's assertion that the child had no loss of hearing.

A clue in this case came when the father said that he never ate a meal away from home, that he would no more eat in public than he would take a bath in public. He asserted, "The girl is just like me."

These illustrations give some idea concerning the embellishments one meets in uncommon forms of psychosomatic manifestations.

In a previous paper I presented a psychological exposé of the subconscious mind in patients who came to me with psychosomatic ear, nose, and throat complaints and who later were analyzed and followed by competent psychiatrists to whom I had referred them. Frankly, until this study came out I never realized how complicated, how serious, and how filthy the subconscious mind could be! No wonder we have psychosomatic disorders!

Weiss and English have asserted that of the patients routinely coming into a physician's office (*a*) one-third have symptoms that are wholly psychosomatic in origin; (*b*) one-third have symptoms that are partially psychosomatic and partially organic in origin, and (*c*) only one-third have symptoms that are wholly organic in nature.

If my calculations are correct, Weiss and English are telling us that nearly one-half of the patients coming into our offices have symptoms that are partially or wholly psychosomatic in nature.

If, then, so many of the complaints coming to a physician's attention have psychosomatic complexions, what greater challenge can a physician have than to acquaint himself with the manner in which both the normal and the abnormal mind works under conditions of quiet and under conditions of stress?

Since all psychosomatic disorders are basically emotional disturbances, we are compelled to consider the importance of understanding what a normal emotion is and how it responds to conditions of quiet and stress and what an abnormal emotion is and how it responds to conditions of quiet and stress.

An emotion we define as "a departure from the normal calm state of an organism of such a nature as to include a strong feeling, an impulse toward an open expression, and certain internal physical reactions." An emotion is any one of the states we designate as fear, anger, disgust, grief, joy, surprise, yearning, etc., and is—when present to an excessive degree—an important agent in the production of psychosomatic disturbance.

I repeat that all psychosomatic disease is basically an emotional disturbance.

(*a*) An expressed emotion we define as a feeling in which an impulse or driving force is directed against the patient's environment, against a set of circumstances which he will not accept, or against counteracting impulses of another person or persons.

(*b*) A repressed emotion we define as a feeling in which two or more impulses or driving forces conflict within an individual's own mind and are antagonistic to each other.

(*a*) An expressed emotion is observed in a woman who had a lump in her throat after the care of her father, who died of a malignant growth of the neck. Dismissing the commonest explanation of a "lump in the throat," namely, a cancerphobia, I found that this particular patient presented a driving force not against her environment—that is, the illness of her father and all the work that it entailed—but against a brother and sister who did not appreciate the sacrifice she had made on her father's—and her brother's and her sister's—behalf. Her somatic reaction to this lack of appreciation was to have a "lump in her throat"—as her father had had—as tangible evidence that in addition to all her physical efforts for the father she now had contracted the disease as well. This is the psychosomatic mechanism by which the brother and sister are expected to pay in terms of remorse a debt resulting from the lack of appreciation of the patient's service and sacrifice for the father and for them.

(*b*) A repressed emotion is observed in the case of the boy working in the tobacconist's shop who had a psychic rhinitis. He presented two antagonistic impulses within his own mind: (1) an urge to continue stealing in order to impress his pals that he had plenty of pocket cash and (2) an urge to discontinue stealing in order to keep from going to the reform school. Another example of a repressed emotion is noted in the case of the woman who had "a terrible pain in her throat." She also presented two antagonistic impulses: (1) an urge to have the cataract removed from the untreated eye, so that she could have good vision in both eyes, and (2) an urge to have a contraindication to the operation by retaining "a terrible pain in her throat."

But why, we ask, does a patient have psychosomatic symptoms at all? For only one reason, namely, he has not learned to make the continual adjustments that life requires. He has not experienced the fun of being tossed about on life's turbulent sea and of taking what comes with a smile no matter what it is. He has not learned the difference between passive and active resistance. He protests when he should ignore. He ascribes to himself responsibilities that he should not assume. He has not learned that doing nothing is often much better than doing something.

The physician must understand that "adjustment implies a constant interaction between the patient and his problems and that each makes demands upon the other. He must know that adjustment is accomplished when the patient yields and accepts conditions which are beyond his power to change, or that it is achieved when environment yields to the patient, or as in most cases that adjustment is a compromise between these two extremes. Maladjustment is a failure to achieve a satisfactory compromise and is the cause of psychosomatic disorder."*

What then are the characteristics of an adjusted personality that should counteract a psychosomatic tendency? I agree with Stanley Gray, who says the following qualities are essential to an adjusted personality:

Happiness (The degree of happiness you experience is an accurate indication of your mental balance); Self-honesty (which is seen in the person who is realistic and who sees things as they are regardless of how they appear); Emotional Control (How often emotions cause a person of superior intelligence to act like a person of inferior intelligence); Enjoyable Occupation (John Dewey said, "If a man is vocationally adjusted and working conditions are as they should be, he can get leisure values out of his labor"); Harmonious Habits and Ideas (Well has it been said that it is mentally unhealthy for an individual to conflict with other people, but it is even worse to conflict with himself), and Physical Health (In my opinion, "good health is almost a prerequisite to a good personality").†

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* Quoted by permission from "The Abnormal Personality" by Robert W. White, Ph.D., New York, Ronald Press Co., 1948.

† Cited by permission from "Psychology in Human Affairs" by J. Stanley Gray, New York, McGraw-Hill Book Company, Inc., 1946.

Progress in Esophagology

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The progress that has taken place in esophagology has been due to several factors. There is a better understanding of the anatomy and physiology of the esophagus. New instruments have been invented. Anesthesiology has progressed, with great benefit to the esophagologist and thoracic surgeon. Improved local and general anesthetic agents are available. The development of chemotherapy and antibiotic therapy has been an important factor in the management of infections associated with endoscopic or surgical procedures on the esophagus. There has been a combined effort by the esophagologist, gastroenterologist, radiologist, and thoracic surgeon to solve the problems of patients who have disease of the esophagus.

In this paper the factors which are thought to represent progress in esophagology in the last decade are discussed. The scope of the subject is so vast that, in many instances, important material can only be mentioned without going into detail.

INTRODUCTION

One of the pleasures of life is to sit down and enjoy the food which is placed before us. Our special senses of sight, smell, and taste give rise to stimuli which, in turn, initiate a variety of responses that are concerned with digestion. The presence of esophageal disease interferes greatly with the enjoyment of food.

Nature requires us to take our nourishment by mouth if life is to be sustained. Medical science has circumvented this requirement by the administration of food by other routes, by gastrostomy, jejunostomy, or intravenous feeding. The latter methods are frequently necessary for patients with esophageal disease. The establishment of continuity as well as good function in the esophagus or its substitute from the pharynx to the stomach is the goal of the esophagologist, gastroenterologist, and surgeon.

The majority of disorders of the esophagus are benign and can be treated successfully by conservative measures. It is an established fact that the surgical correction of diseases of the esophagus may occasion new problems in place of the original disorder. Holinger, Johnston, Potts, and da Cunha [1] and Baker, Flood, and Ferrer [2] have reported on the development of postsurgical strictures. The damaging result of esophageal reflux following surgery for benign conditions has been studied extensively.[3]

ANATOMY AND PHYSIOLOGY

Lerche [4] has written a complete text on the applied anatomy and physiology of the esophagus. On the basis of anatomic studies, the lower end of the esophagus has been divided into the ampulla, which is that segment located just above the diaphragmatic hiatus, and the vestibule, which is the segment connecting the ampulla with the cardiac end of the stomach. This anatomic concept is of importance in relation to some of the newer knowledge of esophageal physiology.

Many of the common esophageal complaints are manifestations of a derangement

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of the motor behavior of the esophagus. A better understanding of the intricate mechanisms which are involved in such disorders would probably result in improved methods of management. At present, this is an area of medicine in which knowledge is still in an early state.

Studies of the disorders of the esophagus afford a relatively simplified approach to some of the mechanisms which control smooth muscle function in the gastrointestinal tract. The esophagus is an organ which is relatively accessible. Its interior can be inspected. By means of the swallowing act, propulsive motility can be readily initiated for study. The esophagus is relatively unencumbered by the presence of secretions from its wall. Continuing investigation of this conveniently situated organ offers the promise of affording a better understanding of motor mechanisms which may ultimately find wider application to some of the problems in esophagology.

The motor activity of the normal esophagus is rather complex. It has been studied by radiologic techniques, by intraluminal balloon recordings of pressure measurements, and recently by direct recordings of intraluminal pressure.* The motor behavior of the upper seven-eighths of the esophagus after the initiation of swallowing is that of a typical propulsive peristaltic wave. Pressure measurements indicate that as the contraction wave travels distally a sharp rise in the intraluminal pressure occurs.

The distal esophagus behaves in a different manner. In the region of the ampulla the pressure increases with peristalsis but falls off more gradually. In the vestibule, according to Ingelfinger, the typical peristaltic wave does not occur.

Disturbances in the motor activity of the esophagus are found in several clinical entities: cardiospasm, insufficiency of the cardia in hiatus hernia, and a newer group, disturbances that follow surgical procedures on the esophagus, particularly those which bypass or ablate the cardiac sphincter. In the last group esophageal reflux may develop and esophagitis or a stricture may form.

Achalasia or idiopathic cardiospasm is characterized physiologically by two disorders in mechanism. There is a tonic contraction of the most distal segment of the esophagus, corresponding approximately to that area defined anatomically as the vestibule. The muscle wall in this region relaxes only infrequently, and dysphagia is the result. In addition, the body of the esophagus in achalasia exhibits a loss of tone. Primary peristalsis fails to occur with the swallowing act.[7] This change is equally characteristic of the disease. Both the tonic contraction in the vestibule region and the changes in the body of the esophagus are probably due to damage to the intramural nervous control of the esophagus.[8]

There has been no striking progress in the management of the patient with cardiospasm. Definitive treatment is directed toward relief of the obstruction resulting from the tight contraction of the cardia and is in a sense palliative. No measures are available for restoring tone and normal peristalsis in the body of the esophagus. Divulsion of the cardia by mechanical means gives the most lasting relief. Mercury bougies are helpful and can be employed safely by the patient. It is now known that surgical correction of cardiospasm should be undertaken only when all conservative methods have failed. The exception is the patient who will not tolerate conservative therapy and insists on a surgical procedure. Ochsner and DeBakey[9] have reported on the surgical treatment of cardiospasm. These authors indicate that there has been no single surgical procedure that helps all patients. A procedure which leaves the gastroesophageal junction intact is to be desired. The Heller type of operation, in some instances, gives a good functioning result, but it can also be followed by a recurrence of symptoms or by esophagitis.†

Insufficiency of the cardia permits the reflux of gastric contents into the lumen of the esophagus. This disorder of the cardiac

* References 5 and 6.

† References 1 and 10.

sphincter may be observed in a variety of conditions. Patients with hiatus hernia constitute numerically one of the most important groups.

The occurrence of esophageal reflux in persons with hiatus hernia may be demonstrated by several techniques.[11] Fluoroscopic examination of the barium-filled stomach in patients with hiatus hernia may demonstrate reflux of barium into the esophagus in approximately 50% of patients. Regurgitation of acid gastric juice into the esophagus is also demonstrable in a high percentage of patients by direct intubation of the esophagus with a small-bore stomach tube. Esophagoscopic findings in the average patient with hiatus hernia support the concept of a relaxed cardia, with the characteristic finding that the instrument passes very easily into the stomach pouch and often regurgitation of fluid back into the esophagus will be noted during the procedure.

The factors which may influence the competency of the so-called sphincteric mechanism at the lower end of the normal esophagus are numerous and are of importance in relation to hiatus hernia. These factors include the degree of acuteness of the esophagogastric angle, which may act as a one-way valve, and the degree of support of the lower end of the esophagus afforded by the crura of the diaphragm. Other factors are the amount of intra-abdominal pressure and the tone and peristalsis of the stomach and lower esophagus.

In the conservative management of patients with hiatus hernia, an attempt to control a few of these factors may be instituted. Such measures as the avoidance of large meals and the avoidance of lying in the completely recumbent position may prove helpful. Weight reduction may often be indicated. Permanent relief, however, usually can be obtained only through proper surgical intervention, if and when the degree of disability justifies such a procedure.

In simple heartburn, there is also evidence that suggests that incompetence of the cardia may be important, at least in some cases.[12] In a series of patients with heartburn, but with no hiatus hernia, regurgitation of gastric juice during esophageal intubation was encountered in approximately one-half of the patients studied. However, radiologic studies revealed reflux in only a small percentage of these persons.

The occurrence of inflammatory changes in the lower end of the esophagus in many patients with hiatus hernia has long been recognized.[13] Only in recent years has the association of esophagitis with hiatus hernia become generally appreciated. An important factor has been the introduction of the flexible esophagoscope, rendering endoscopic examination of the lower esophagus a procedure which is safe and more generally available.

At present, most workers favor the view that the inflammatory changes in the lower esophagus in patients with hiatus hernia are due to the irritating effects of regurgitated gastric juice upon the squamous lining of the esophagus. It should be noted, however, that the evidence for this concept is not as yet conclusive. Reflux can, for example, be demonstrated in many persons who present no gross evidence of esophagitis. Of interest in this connection is the observation of Palmer,[14] who found histologic evidence of esophagitis on biopsy in many patients with hiatus hernia subjected to examination.

Further support of present theories of peptic esophagitis may be found in persons who for one reason or another have undergone a surgical procedure which bypasses or ablates the cardiac sphincter. Several observers have reported on changes found in the esophagus after resection of the cardiac sphincter with esophagogastrostomy and/or other surgical procedures. Esophagitis and esophageal reflux are frequently demonstrated in such postsurgical patients. Of special interest is the occasional patient who has undergone a total gastrectomy and develops esophagitis. In this situation, the cardiac mechanism is destroyed. Alkaline contents from the small bowel flow freely back into the esophagus. It may be assumed

that the esophagitis is attributable to surface irritation by the small bowel contents, a "tryptic" rather than a "peptic" esophagitis.

The problems of reflux and resultant esophagitis should always be considered when operative procedures are undertaken in the lower esophagus. Too often the patient develops esophagitis after operation.

ANESTHESIA

The anesthesiologist has been of great help to the esophagologist and thoracic surgeon. The combination of a local anesthetic, either 2% cocaine, 0.5% tetracaine (Pontocaine) hydrochloride, or 2% lidocaine (Xylocaine) hydrochloride, with intravenous thiopental (Pentothal) sodium is very satisfactory for esophagoscopy and gastroscopy. The local anesthetic agent is sprayed in the pharynx and also instilled into the larynx until the cough reflex is abolished. At this point, intravenous thiopental sodium is administered in sufficient dose that the patient just loses consciousness. The esophagoscope is then introduced when the patient is breathing in regular rhythm. The local anesthetic agent is necessary to prevent laryngeal spasm.

To the thoracic surgeon, the anesthesiologist has been most helpful. A better understanding of the physiologic mechanisms has brought about a marked reduction in operative mortality. Also newer anesthetic agents are constantly being developed to solve the problems of the thoracic surgeon.

NEWER INSTRUMENTS

The flexible esophagoscope (Eder-Hufford [15]) has been a worth-while addition to our instrumentarium. This tube has a flexible obturator which is introduced into the esophagus in the same manner as the flexible gastroscope. After the obturator has entered the upper third of the esophagus, the esophagoscope is passed over it through the cricopharyngeus into the esophagus, and then the obturator is withdrawn. This esophagoscope can be used on the average outpatient without difficulty after a local anesthetic agent has been applied to the pharynx. It should not be employed for lesions of the upper third of the esophagus. It has its greatest value in diseases of the lower esophagus. The Eder-Hufford tube has been used in our outpatient gastroenterology department for the past four years without an accident.

The use of magnets for extraction of magnetizable foreign bodies in the esophagus and stomach represents an advance in esophagology. Equen [16] has called our attention to this effective procedure.

The telescope introduced through the esophagoscope allows for a more thorough study of the mucous membrane or of specific lesions at the distal end of the esophagoscope.

Inflation of the lower esophagus is helpful in the study of disease at this level. Putney and Clerf [17] found it useful in their study of epiphrenic diverticulum.

A combination of esophagoscopy and flexible tube gastroscopy with inflation of the lower esophagus is helpful in the study of hiatus hernia.

CORROSIVE BURNS OF THE ESOPHAGUS

Stricture formation in the esophagus due to the ingestion of corrosive agents is a never-ending problem to the esophagologist. Prophylactic measures against stricture formation include early intraesophageal manipulation.[18] The latter may be attended with serious dangers, and great care is required in performing mechanical treatment and examination of the injured esophagus. The Salzer technique and early bougienage are the methods of choice in preventing stricture formation. Antibiotics have helped greatly in controlling the infection associated with this type of injury.

Cortisone therapy has received a great deal of attention. This type of therapy prevents fibroplasia and might be expected to inhibit stricture formation by preventing collagenization. Smith, Compton, and Palmer [19] have suggested that cortisone administration should be started immediately after injury has occurred. The treatment is potentially dangerous, and all patients should

be closely supervised in a hospital and given liberal amounts of antibiotics.

The investigations of Rosenberg, Kunderman, Vroman, and Moolten [20] show that further study of cortisone therapy is necessary before it can safely be employed in acute lye burns of the esophagus. They found a high incidence of perforation of the ulcerated esophagus in rabbits under cortisone therapy.

Esophagoscopy in the early stages of corrosive burns of the esophagus should be performed with great care. It should always be done within 10 days of the injury to determine the extent of involvement of the mucous membrane.

MISCELLANEOUS DISORDERS

Deficiency diseases affect the esophagus. Plummer-Vinson syndrome with web formation at the cricopharyngeus continues to be a challenge. The web is easily dilated, but the esophagitis is difficult to treat. Therapy of the iron deficiency remains a problem. This disease has become increasingly rare in recent years.

The development of esophageal varices and the management of the diseases which may favor the formation of varices are broad subjects beyond the scope of this presentation. Some of the newer procedures for the control of hemorrhage from varices deserve special mention. The use of the Sengstaken ballon [21] has, in our experience, been a lifesaving measure in many instances. Only occasionally is direct surgical intervention for the control of esophageal bleeding necessary or advisable. Opinion continues to vary concerning the usefulness of injection therapy for varicosities of the esophagus. At all events, the development of various shunting procedures has made available more definitive methods of treatment for esophageal varicosities.

ESOPHAGEAL SURGERY

The surgical technique for esophageal disease is beyond the scope of this paper. Progress in esophagology, however, has been in a large measure due to the advances in the field of thoracic surgery.

Operations can be performed skillfully and without fear of mortality. Congenital tracheoesophageal fistula and atresia, as well as stenosis of the esophagus due to a vascular ring, have been conquered by the surgeon.

Although the surgeon may eradicate the stenosis or disease, the patient may continue to have difficulty with the esophagus. A stricture may re-form, or reflux with esophagitis may develop. These are problems for the esophagologist and gastroenterologist.

Carcinoma of the esophagus is not a challenge from the surgical standpoint. The problem is that of making an early diagnosis before the disease has spread either locally or by metastasis. The survival rate at the end of five years is very low.

The one-stage operation for pulsion diverticulum of the hypopharynx has now been widely adopted.

SUMMARY

Progress in esophagology has been the result of the continued efforts of all branches of the medical sciences, including radiology, anesthesiology, anatomy, and physiology.

The esophagologist, gastroenterologist, and thoracic surgeon are responsible for the treatment of patients with esophageal disease.

The problems that are encountered in esophageal disease are many and varied.

Conservative therapy should always be carried out until it is established that a functional esophagus cannot be obtained. If conservative therapy fails, then surgery should be employed. Postsurgical problems develop in many patients and have to be treated by the esophagologist and gastroenterologist.

Esophagology includes the work of the radiologist, thoracic surgeon, gastroenterologist, and esophagoscopist.

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News and Comment

ANNOUNCEMENTS

Postgraduate Conference in Otorhinolaryngology.—A Postgraduate Conference in Otorhinolaryngology will be presented by the Division of Otorhinolaryngology, Stanford University School of Medicine, March 26-30, 1956. This Conference will offer a comprehensive survey of the current field of ear, nose, and throat. Presentations will be didactic, by individuals and panel groups. Some of the subjects will be basic science reviews, such as anatomy (using three-dimensional projection) and pathology; audiology, including Psychogalvanic Skin Response; ear surgery, including stapes mobilization; nose and sinus problems, including rhinoplasty; pituitary surgery; allergy and steroid therapy; throat diseases, including suspension laryngoscopy, laryngoplasty (e. g., use of tantalum plate), and laryngectomy with neck dissection; speech problems; bronchoscopy and esophagoscopy; etc.

Registration is limited to 30 Doctors of Medicine in E. N. T. or E. E. N. T., and the fee is $100.00. Application may be made to Office of the Dean, Stanford School of Medicine, 2398 Sacramento St., San Francisco 15.

Audiology Section

JOSEPH SATALOFF, ASSOCIATE EDITOR

Hearing and Deafness in Cleft-Palate Patients

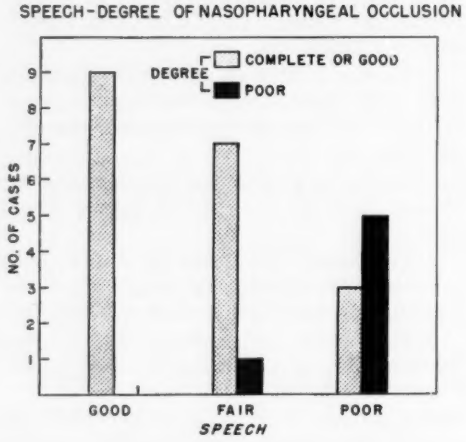
EDGAR M. HOLMES, M.D.
and
GEORGE F. REED, M.D., Boston

It has long been known to otologists that the incidence of deafness and ear infections in patients with cleft palates is high. Notwithstanding the importance of this subject, a study of the literature revealed but few articles on the problem. One enlightening article by Sataloff and Fraser [1] was found in which 30 cases were reviewed. In this series, only three persons had normal hearing. All the remaining cases showed significant hearing loss and ear pathology. In view of their findings and the dearth of information dealing with the subject, we decided it might be helpful to evaluate the cases from the Massachusetts Eye and Ear Infirmary and the Massachusetts General Hospital which had been operated on during the past 10 years. Requests were sent to 112 cases to report for this evaluation. Unfortunately, cooperation was not too good, so that we were able to thoroughly study and evaluate only 26 of the 112.

On each patient a thorough history was taken and a careful ear, nose, and throat examination was performed, including an estimate of the postoperative palate as to whether it was under tension from scarring or insufficient surgical relaxation of the lateral attachments or was of nearly normal flexibility and appearance. An estimate was also made regarding nasopharyngeal occlusion, being graded complete, good, or poor.

Accepted for publication July 28, 1955.

From the Department of Plastic Surgery, Massachusetts Eye and Ear Infirmary.

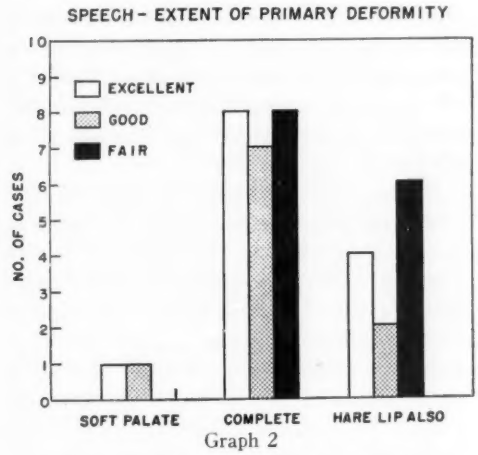


Graph 1

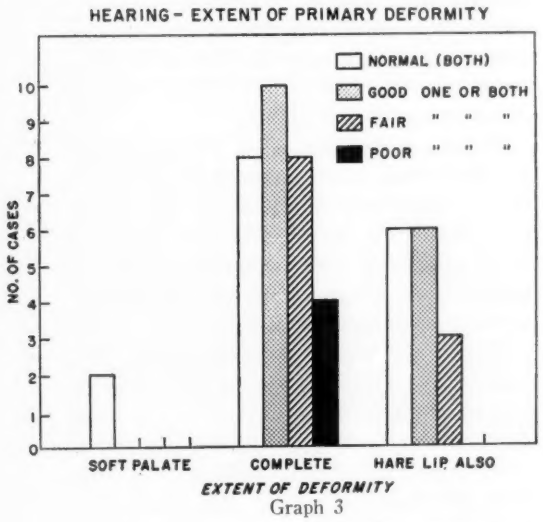
The nasopharynx was examined, where possible, and the Eustachian tubes were inflated in all cases. The ears were examined thoroughly. The throat was inspected for the presence or absence of tonsils, and an air-and-bone audiogram was then performed on each patient.

The total group consisted of 26 cases, 24 of whom had had a complete cleft palate. In two it was limited to the soft palate. Of the total group, 12 also had a harelip, 7 of which were unilateral and 5 bilateral.

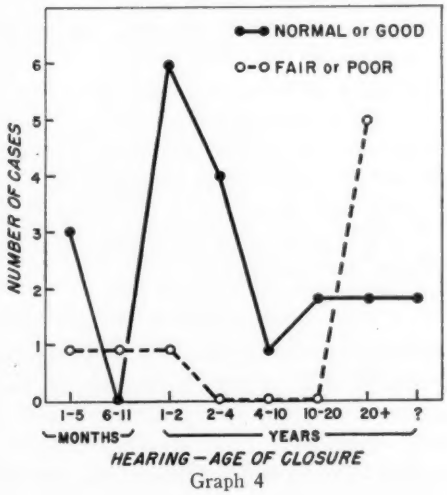
We were not primarily concerned with speech in these patients, but, since they were being studied, we arbitrarily graded their speech as good, fair, or poor and compared speech against other factors resulting from our evaluation for hearing. Good speech was considered as understandable with slight nasality. Fair speech included those with moderate nasality and noticeable speech abnormality. Poor speech included those with pronounced difficulty in speech production. As might be expected, this showed



Graph 2



Graph 3



Graph 4

a very definite and direct relationship between the quality of speech and the degree of nasopharyngeal occlusion (Graph 1). It was also found that there was no relation between the age of closure and resultant speech, nor was there any connection between the speech and the degree of primary deformity (Graph 2).

HEARING EVALUATION

Hearing was classified into four groups: normal, 0 to 10 db. in both ears; good, 10 to 20 db. in one or both ears; fair, 20 to 35 db. in one or both ears; poor, below 35 db. in one or both ears. Decibel level was obtained by averaging 500, 1000, and 2000 frequencies.

Of the total group of 26 persons, only 10, or 38%, had normal hearing; 16, or 62%, had a hearing loss greater than 10 db. in one or both ears, and of this 16, 12, or 46%, of the total had a hearing loss greater than 20 db. in one or both ears. A statistical breakdown was then made on all cases in order to compare their hearing with other factors. In dealing with these figures, we realize that the numbers are rather small to be of real statistical significance and the statistics and graphs, therefore, should not be taken too literally. We do feel, however, that our conclusions are honest and accurate despite the small number of cases. The statistics and graphs are presented with this understanding, so that the reader can see on what basis we have formed our impressions.

Graph 3 shows no definite relationship between the severity of primary deformity and hearing.

Graph 4 shows no definite relationship between hearing and age of closure, although there does seem to be a tendency indicating that the earlier the closure is performed the better the hearing will be.

Table 1 shows no relationship between hearing and the degree of tightness or flexibility of the postoperative palate.

Although the nasopharynx could not be seen in all patients, there was no relation

TABLE 1.—*Relationship Between Hearing and the Degree of Tightness or Flexibility of the Postoperative Palate*

Hearing	Flexible Palate, %		Tight Palate, %	
Normal	26	61	50	75
Good	35		25	
Fair	26	39	13	25
Poor	13		12	

TABLE 2.—*Relationship Between Ear Pathology and the Presence or Absence of Tonsils*

Ear Pathology	Tonsils Present		Tonsils Absent	
	No.	%	No.	%
Normal	12	30	4	33
Scarred and retracted	23	57	6	50
Perforation	4	10	2	17
Microtia	1	3	0	0

TABLE 3.—*Relationship Between Hearing and the Presence or Absence of Tonsils*

Hearing	Tonsils Present		Tonsils Absent	
	No.	%	No.	%
Normal	17	42	7	58
Good	10	25	4	33
Fair	8	20	1	9
Poor	4	10	0	0

TABLE 4.—*Number of Upper Respiratory Infections After Closure of Cleft Palate*

	No.	%
More	4	15
Same	8	31
Fewer	9	35
Unknown	4	15
Not closed (obturator)	1	4

between the hearing and the presence or absence of adenoid tissue in those patients in whom it was possible to examine the nasopharynx.

Regarding the examination of the ears, 8 of the total 26, or 38%, showed normal tympanic membranes. All the others, or 62% of the total, showed pathology consisting of either scarring, retraction, or healed perforations; 6, or 24% of the total, showed perforations in one or both ears. We were unable to demonstrate any relationship between the ear pathology and presence of tonsils (Table 2), nor could we demonstrate any relation between the age of closure and the amount of ear pathology.

The presence or absence of tonsils in relation to the hearing loss interested us greatly, and the group showed that 20 of the 26 had tonsils present on examination and 6 showed absent tonsils. Comparing the presence or absence of tonsils against hearing seemed to indicate a tendency toward better hearing in those patients who have had a tonsillectomy (Table 3). Adenoids were evaluated in all but five cases, and no relationship could be shown between hearing and the presence or absence of a significant amount of adenoid tissue, nor was there any relation between adenoids and speech quality.

Closure of the cleft palate did not appear to have much effect upon the number of upper respiratory infections or attacks of otitis media. As shown in Table 4, 35% had fewer attacks of upper respiratory infection after closure, whereas 46% had the same or more attacks following closure.

Although one cannot demonstrate it with figures or statistics, we both were very much impressed with the disproportion between the amount of pathology that we saw in the drum membrane and the amount of hearing loss. In nearly all cases, after examining the eardrum, one would expect to find a much greater hearing loss than was found by audiometry.

Of the total group of 26 persons, 12 gave a history of having had no known attacks of otitis media; 6 of the 12, or 50%, however, showed definite evidence on examination of scarring and retraction of the drum, indicating that they had had previous infections without their knowledge.

COMMENT

When one realizes from the above figures that 62% of all patients have a hearing loss greater than 10 db., the problem becomes a very serious one. It becomes especially important to study and understand this hearing

loss, since the loss, as seen in these patients, is of a permanent, untreatable nature. The losses were all of the conductive type and due to chronic middle ear changes in the form of chronic disease from perforations or from fibrosis and adhesions in the middle ear; therefore, these losses are not remedial, and it becomes most important to determine how the loss can be prevented. One can assume from the above figures that the problem is not to be solved by changing the present concepts regarding closure of the cleft palate, since the figures show no relation between hearing and the various factors relative to the severity of the cleft palate; also, there seems to be no real evidence that the presence or absence of tonsils and adenoids has much effect on the hearing or amount of pathology in the middle ear. What then is the cause? It would seem reasonable to assume that these patients' ears are subjected to many and repeated infections. It is not difficult to visualize the amount of trauma and infection that the Eustachian tubes are subjected to in these cases. With a patent palate, each time the person swallows, mouth contents are driven upward toward the nasopharynx by the tongue carrying potentially infected food past, if not into, the Eustachian tubes. If this is to be considered the cause of chronic ear infection in these people, the natural assumption would then be, and has been by many in the past, that the answer to preventing hearing loss would lie in early closure of the cleft palate. Unfortunately, our 26 cases do not bear this out. Apparently, even after closure or prior to closure at an early age, the nasopharynx is subjected to more trauma or pathology and infection than is the nasopharynx of normal children. Perhaps it is best that our figures do not indicate an early closure, because some surgeons prefer to delay the closure of the palate until the child has reached 2 years of age, feeling that the result is better if the child is older and one is able to use a plastic splint over the repaired palate during the healing period.

Regarding the removal of the tonsils and adenoids, our results in these cases do not indicate any definite advantage in the patients who have had a tonsillectomy and adenoidectomy; therefore, on the basis of these cases, at least, we cannot fairly make any assumptions as to the advisability of a tonsillectomy and adenoidectomy in reference to prevention of hearing loss. It is probably still better to consider each case as a separate entity in determining the advisability of a tonsillectomy and adenoidectomy, such a procedure being carried out only after complete agreement between the pediatrician, the otologist, and the plastic surgeon, with great pains being taken to carefully preserve all possible normal tissue in the pharynx for the preservation of the best possible speech.

What then can be done to prevent this hearing loss? As we see it, after reviewing these cases, the only practical control which we have over repeated infections of the middle ear, with resultant hearing loss, is more careful detection and observation of the ear findings in these persons.

As has been pointed out, approximately 50% of those people giving no history of ear infection showed rather marked tympanic membrane findings, indicative of previous infections. Since the hearing loss is resultant from repeated middle ear infection, probably of minor nature at the time, with resultant scarring, fibrosis, and fixation of the middle ear structures, the best prevention of chronic ear changes lies in rapid recognition of the disease process and prompt treatment before there is a chance for organization of undrained, purulent, or serous debris in the middle ear; therefore, we feel that it is most important that parents be cautioned at the outset of the great necessity of reporting all minor ear symptoms to the otologist as soon as they appear. Fortunately, today, with antibiotics and chemotherapy, such acute infections can be fairly easily controlled at the time of the acute infection, whereas the later process cannot be altered.

SUMMARY

In a series of 26 cleft-palate patients, 62% were found to have a hearing loss greater

than 10 db. in one or both ears. No relationship could be found between this hearing loss and the age of closure of the palate, the degree of flexibility of the closed palate, the amount of nasopharyngeal occlusion, or the severity of tne initial deformity, nor could any relationship be shown between the hearing loss and the presence or absence of tonsils or the presence or absence of an abnormal amount of adenoid tissue. The only practical means of controlling and preventing this hearing loss is that of prompt recognition and early treatment of all ear infections in this group of patients. A plea is made for parents and physicians concerned with these patients to be ever aware of the great importance of early treatment of even minor ear infections.

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Locus of the Lesion in Recruiting Ears

PHILLIP A. YANTIS, Ph.D., Ann Arbor, Mich.

The place of loudness recruitment testing in otological diagnosis has been recognized for more than 10 years. However, until recently, the diagnostic value of such tests was limited to the differentiation of conductive and perceptive types of hearing losses. The value of these procedures with respect to the further delineation of the anatomical locus of the lesion in perceptive losses was not realized.

Accepted for publication July 18, 1955.

From the Physiological Acoustics Laboratory, Institute of Industrial Health, University of Michigan. This study was undertaken in preparation for a doctoral dissertation under the supervision of Dr. Merle Lawrence and Dr. George Herman, of the University of Michigan, and was supported in part by the Research and Development Division, Office of the Surgeon General, Department of the Army, under Contract DA-49-007-MD-634.

With the recent publication of recruitment studies on abnormal ears of various types, the diagnostic value of recruitment testing was increased significantly. De Bruïne-Altes[8] was one of the first to include enough varieties of verified causative classifications to make general delineating conclusions; however, there was still a need for data on persons with eighth nerve lesions. Dix, Hallpike, and Hood [7] were among the first to supply such information and to call attention to the value of recruitment testing in differentiating cochlear and noncochlear lesions in perceptive type hearing losses. More recent research has continually added further data on increasingly larger varieties of losses.

The accumulated evidence has appeared sufficiently consistent to make the established existence of recruitment a reliable aid in verifying the locus of the lesion causing perceptive losses. Therefore, the published information on the results of recruitment

TABLE 1.—*Loudness Recruitment Tests That Have Been Proposed in the Literature, with a Brief Identifying Description of Each*

Test No.	Authors	Year	Reference No.	Description of Test
1	Fowler	1936	11	"Alternate binaural loudness balance"
2	Reger	1936	30	"Alternate monaural loudness balance"
3	Watson and Knudsen	1940	34	"Auditory span" between thresholds of audibility and feeling at 1 frequency
4	Huizing	1942	18	"Monaural masking"—a masking and a masked tone impressed on same ear
5	Huizing	1942	19	"Index vocalis"—comparison of distance at which whispered and spoken voices are just audible
6	Békésy	1947	2	"Békésy audiometer"—difference limen (DL) at threshold levels for continuous frequency range
7	Lüscher and Zwislocki	1948	28	DL at 40 db. above threshold at 1 frequency
8	Watson and Tolan	1949	33	"Most comfortable loudness level" for pure tones
	Kobrak	1950	22	"Loudness shock phenomenon"—tuning fork of high frequency struck at maximum power
	Denes and Naunton	1950	5	DL of 2 sounds of same frequency presented alternately at 4 and 44 db. above threshold
9	Jerger	1952	21	"Northwestern DL"—DL difference of tone of 1 frequency presented 10 and 40 db. above threshold

TABLE 2.—*Summary of Results of Recruitment Testing in 592 Cases with Abnormal Hearing Reported in the Literature*

Locus of Lesion and Diagnosis	Cases	Recruiting	Not Recruiting	Indefinite	Tests Used*	Reference No.
Group I: Conductive (Outer or Middle Ear)						
Artificially blocked external auditory meatus	1	0	1	0	7	26, 28
	2	0	2	0	†	3
	2	0	2	0	1	16
Atresia of external auditory meatus	1	0	1	0	†	3
Perforated tympanic membrane	1	0	1	0	†	3
	1	0	1	0	8	21
Eustachian tube congestion	1	0	1	0	†	3
Eustachian tube stenosis	1	0	1	0	7	26, 28
Otitis media	5	0	5	0	8	21
Acute suppurative otitis media	1	0	1	0	†	3
	3	0	3	0	7	26
Acute hemorrhagic aero-otitis media	1	0	1	0	1	16
Residue, acute otitis media	1	0	1	0	†	3
Chronic otitis media	2	0	2	0	†	3
	5	0	5	0	7	26
	1	0	1	0	7	28
Otosclerosis, normal bone conduction	2	0	2	0	1, 2	17
	7	0	7	0	8	21
	27	0	27	0	1	25
	1	0	1	0	7	26
	1	0	1	0	7	28
Totals, Group I	78	0	78	0		
Group II: Mixed (Middle and Inner Ear)						
Otosclerosis, decreased bone conduction	2	0	0	2	†	3
	3	0	3	0	1, 2	17
	13	13	0	0	8	21
Radical mastoidectomy, decreased bone conduction	1	0	0	1	†	3
Otitis media, decreased bone conduction	4	4	0	0	8	21
Totals, Group II	23	17	3	3		
Group III: Perceptive (Cochlea)						
Ménière's disease	15	15	0	0	†	3
	4	4	0	0	1	6
	30	30	0	0	1	7
	30	30	0	0	1	8
	2	2	0	0	1	13
	50	42	8	0	?	15
	13	13	0	0	1, 2	17
	6	6	0	0	8	21
	1	1	0	0	1, 4	23
	26	26	0	0	6	24
	20	20	0	0	1	25
	6	6	0	0	7	26
	1	1	0	0	7	29
Ménière's disease (32 cases) and vascular accident involving cochlea (1 case)	33	31	0	2	1	9
Acoustic trauma	30	30	0	0	†	3
	1	1	0	0	4	4
	2	2	0	0	1	16
	3	3	0	0	8	21
	25	25	0	0	6	24
	14	14	0	0	7	26
Explosion trauma	7	7	0	0	†	3
Labyrinthine concussion	3	3	0	0	†	3
	9	3	3	3	7	26
Totals, Group III	333	317	11	5		
Group IV: Perceptive (Eighth Nerve and Central Lesions)						
General Trauma						
Nerve trauma	1	0	1	0	1, 2	17
Brain damage	1	0	1	0	8	21
Head injury						
Temporal region, no fracture	1	0	1	0	1	31
Linear fracture, cranial fossa	1	0	1	0	1	31
Parietal and occipital fracture into foramen magnum	1	0	1	0	1	31
Brain stem lesion	1	0	1	0	6	24
Vascular Accident						
Involving 8th nerve	1	0	1	0	1	9
Thrombosis of anterior-inferior cerebellar or basilar artery	1	0	0	1	1	9
Congenital						
Eighth nerve involvement	1	0	1	0	1, 2	17
Platybasia	1	0	1	0	6	24

TABLE 2.—*Summary of Results of Recruitment Testing in 592 Cases with Abnormal Hearing Reported in the Literature—Continued*

Locus of Lesion and Diagnosis	Cases	Recruiting	Not Recruiting	Indefinite	Tests Used*	Reference No.
Disease						
Parkinson's, with 8th nerve damage by disturbed blood supply	1	0	0	1	1	9
Paget's of the skull	1	0	1	0	1	9
Meningitis	1	0	1	0	1	9
	1	0	1	0	6	24
Pneumococcic meningitis	1	0	1	0	1	9
Encephalitis	1	0	1	0	6	24
Atrophy						
Hemiatrophia cranii	1	0	1	0	6	24
Tabes	1	0	1	0	6	24
Tumor						
Meningioma						
In cerebellopontine angle	2	0	2	0	6	24
Of clivus	1	0	1	0	6	24
Of cranial fossa	1	0	1	0	6	24
Papilloma of 4th ventricle	1	0	1	0	6	24
Metastatic tumor of pons	1	0	1	0	6	24
Brain tumor with 8th nerve lesion	1	0	1	0	1	9
Intracerebral hematoma, secondary to arteriovenous fistula or angioma	1	0	1	0	1	9
Astrocytoma of temperoparietal lobe	1	0	1	0	1	9
Tumor of acoustic nerve or angle lesions	15	0	15	0	1	8
Acoustic tumor	4	1‡	3	0	1	6
	1	0	0	1	1	9
	2	0	2	0	1, 2	17
	9	0	6	3	6	24
	1	0	1	0	6	14
Neurofibroma	3	0	3	0	1	9
Neurofibroma (11 cases) and other varieties of cerebellopontine angle lesions (9 cases)	20	0	14	6	1	7
Acoustic neurinoma	3	0	3	0	1, 4	23
Neuroma	1	1	0	0	1	10
	1	1	0	0	1	12
Cerebellopontine angle tumor	3	0	3	0	1	9
	3	3	0	0	1	‖
	1	0	1	0	8	21
	2	0	2	0	7	26
Eighth nerve neurilemmoma	2	0	2	0	1	32
Totals, Group IV	98	6	80	12		
Group V: Perceptive (Locus Questionable)						
Progressive hereditary deafness	10	10	0	0	†	3
	1	1	0	0	4	4
	1	1	0	0	1	16
	1	1	0	0	8	21
Meningitis	2	2	0	0	8	21
Epidemic cerebrospinal meningitis	2	2	0	0	†	3
Scarlet fever	2	2	0	0	8	21
Syphilis	1	1	0	0	†	3
Congenital syphilis	1	1	0	0	1, 2	17
Presbycusis	4	0	4	0	†	3
	1	0	1	0	4	4
	5, 5§	2	8	0	8	21
Presbycusis, early Ménière's disease	2	2	0	0	1, 2	17
Basal skull fracture	6	2	0	4	†	3
Skull trauma	1	1	0	0	1	16
	1	1	0	0	1, 2	17
	4	0	4	0	1, 4	23
Explosion trauma with commotio labyrinthi	1	0	1	0	†	3
Ménière's disease and allergy	1	1	0	0	1, 2	17
Thrombosis of internal auditory artery	1	1	0	0	8	21
Streptomycin toxicity	1	0	1	0	8	21
	1	0	1	0	7	26
Cochlear neuritis	3	0	3	0	1, 3	23
Mumps	1	0	1	0	7	26
Measles	1	1	0	0	6	14
Totals, Group V	60	32	24	4		

* The numbers refer to the tests listed in Table 1.
† de Bruine-Altes used Tests 1, 2, 3, 4, and 5 throughout her study.
‡ Refer to text for Dix, Hallpike, and Hood's explanation for the presence of recruitment in this case.
§ Presence of presbycusis "probable" in these cases.
‖ Fowler, E. P., Jr., in discussion on Reger and Kos, p. 821.[14]

testing in patients with various types of hearing impairment has been gathered together and is presented here.

DESCRIPTION OF TABLES

Table 1 presents a very brief description of the various tests for recruitment that have been proposed in the literature; a complete description of each procedure may be found in the reference cited for the respective tests. No attempt is made to evaluate these tests here; when the test was employed in a published study cited in this paper, its validity as an indicator of loudness recruitment was not questioned. Some investigators have tended to dispute the validity of certain procedures [25] and have compared the practicality and clinical usefulness of a large number of them.*

Those tests that are numbered in Table 1 were used by one or more investigators in their research and are identified in Table 2 under the title "Tests Used." The remaining procedures have not been employed in published studies on the presence of recruitment in various pathological cases.

Table 2 is a summary of the data reported in the literature concerning loudness recruitment studies on abnormal ears. Cases reported in Groups I to IV are those in which the authors did not seem to question the locus of the lesion causing the loss (outer or middle ear, cochlea, or eighth nerve) or the cause of the loss. No cases of questionable causative factors were listed in this table unless otherwise noted. The cases reported in Group V are those in which the locus of the lesion was either not stated or was questioned by the author concerned. Thus, some writers mentioned particular causes as being eighth nerve lesions, and other investigators were not definitive.

Although some persons may have had a bilateral loss with subsequent testing of both ears, all of the cases reported in any group are the number of patients, not the number of ears, tested. Whenever possible, the etiologic terms used by the respective authors were employed. It may have been possible to combine some classifications, but they were listed separately so as to avoid confusion. However, in Group IV, the classes have been grouped to point out similarity when present. Even so, some overlapping may be found in the medical definitions of these terms but not in the number of cases reported.

Finally, it will be noted that the evidence pertaining to the presence or absence of loudness recruitment is classified under three titles: number recruiting (partial or complete), number not recruiting, and number indefinite. Because most writers do not attempt to quantify the amount or degree of recruitment present in certain cases, and others speak in terms of "partial" and "incomplete" recruitment, all such cases were classified under the category of "recruitment present." The "indefinite" category includes those instances in which the author was not sufficiently clear in his description of individual results.

COMMENT

The data listed in Table 2 were categorized into five groups according to the locus of the lesion in each case. The present discussion will cover each of these groups in turn.

Group I: Conductive Losses (Lesion in Outer or Middle Ear)

There have been no cases of recruitment reported in the literature in cases with a hearing loss due to pathological processes in the outer or middle ear and with normal bone-conduction responses. This evidence is not new and requires no discussion.

Group II: Mixed Losses (Lesion in Middle Ear, with Decreased Bone Conduction)

Most writers have found evidence of recruitment, either partial or complete, in persons having a decreased bone-conduction response but with primary pathology in the middle ear. Assuming recruitment to be a cochlear phenomenon, this would support the conclusion that a secondary perceptive loss of this type is a cochlear involvement.

* References 1, 5, 17, 20, and 27.

Group III: Perceptive Losses (Cochlear Lesion)

The evidence is overwhelming that in cases of a loss caused by at least two disorders known to be cochlear (Ménière's disease and acoustic trauma) marked recruitment is present. The only exceptions to this seem to be three cases of "labyrinthine concussion" reported by Lüscher and eight cases of Ménière's disease mentioned by Hahn. These results are in a definite minority, however, and cannot be adequately evaluated by the independent observer.

Group IV: Perceptive Losses (Eighth Nerve and/or Central Lesions)

The obvious conclusion made from evidence presented in this group is that recruitment is not pathognomonic of central nervous system lesions. Dix, Hallpike, and Hood [7] found one case of a loss due to an acoustic tumor which exhibited recruitment. They mentioned, however, that the possible explanation for involvement of the organ of Corti in this case was a secondary involvement of cochlear blood supply by tumor pressure. Fowler Sr.† is another investigator claiming recruitment to be present in noncochlear perceptive losses as well as in the end-organ.

There may be an obvious, and sometimes overlooked, explanation for evidence of recruitment in cases of eighth nerve pathology, even when verified by operation or by autopsy. Studies conducted on a man 52 years of age with a bilateral high-frequency loss may serve to indicate this oversight. This patient had noticed a hearing loss to a slight degree in both ears for 10 years and a steady decrease in sensitivity in the right ear for two months before admission to the University Hospital. He had been working for 20 years in a noisy industrial environment. Upon examination, he was found to have partial recruitment in the right ear at 1000 cps, using the alternate binaural loudness balance test with masking of the left ear. Partial recruitment was also found in the left ear at 2000 cps, employing the alternate monaural loudness balance test.

The otological diagnosis made was acoustic trauma in the left ear and acoustic neuroma in the right. The diagnosis of the tumor was verified by surgery two days after testing; the growth was situated in the internal auditory meatus. Thus, this case showed partial recruitment in an ear with an apparently noncochlear involvement due to an acoustic neuroma. However, when it is remembered that the patient had a bilateral loss as long as 10 years before, and had been working for a considerable length of time in a noisy environment, it could well be surmised that some cochlear damage due to acoustic trauma may have been present in the right ear before the eighth nerve involvement, thus accounting for the presence of recruitment. Some caution may obviously be necessary in making conclusive judgments without taking into account the possibility of more than one causative factor. This is especially true in cases of a hearing loss existing over a long period of time.

Group V: Perceptive Losses (Locus of Lesion Questionable)

On the basis of the evidence presented in Groups I, III, and IV, there seems little reason to doubt that the phenomenon of loudness recruitment is related closely to the presence of cochlear lesions. The value of this conclusion is obvious in otological diagnosis. But the need for further recruitment studies on pathological ears due to various causes which are not well localized with respect to the anatomical region of affection cannot be underestimated. The evidence presented in this group is not sufficient at the present to draw definite conclusions as to the locus of the lesion in the various causes of perceptive losses listed. However, in some cases, it may be indicative, and the use of loudness recruitment testing in further research with such causes should be of value.

SUMMARY

Published evidence on the presence of loudness recruitment in ears with abnormal hearing due to various causes is classified

† References 10 to 13.

into five groups according to the locus of the responsible lesion. The number of patients tested by each author in a particular classification as to cause is presented, with a breakdown of the number of patients exhibiting the phenomenon or not. The type of test procedure(s) used by each author is also listed.

The evidence presented in each group is briefly discussed, together with a possible explanation for the few discrepancies that exist. The overwhelming evidence in the literature supports the conclusion that loudness recruitment occurs only in lesions involving the sensory end-organ, and is not expected to be present in lesions involving only the eighth cranial nerve and the higher acoustic pathways.

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Speech Audiometry by Bone Conduction

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The importance of the use of speech audiometry by air conduction in conjunction with pure tone tests in the diagnosis and evaluation of hypacusis is unquestioned in the field of otology. Each method contributes uniquely to a fuller understanding of a particular case of hearing impairment. In addition, past research has clearly indicated that a high degree of relationship exists between the binaural speech reception threshold as determined with Harvard spondees and the better ear average threshold value for pure tones 500, 1000, and 2000 cps when both measures are obtained by air conduction. Carhart,* in a series of clinical studies, reported a coefficient of correlation of about 0.75 between the two types of tests for both normal-hearing and deaf subjects. The correlation between the average pure tone threshold and the speech reception threshold is of the same order of magnitude as the test-retest reliability of either method.

Pure tone thresholds by bone conduction audiometry have likewise been shown to be almost as reliable as by air conduction when adequate precautions in testing are observed.† Also, there appears to be no appreciable difference in the results obtained for the 250 to 4000 cps range with either a small hearing aid or plunger type of bone receiver.‡

Although the literature is relatively replete with studies pertaining to acuity tests for speech and pure tones by both air and bone conduction, no attention, so far as we are aware, has been directed toward the use of a speech reception threshold by bone conduction.

The purpose of the present study was to investigate the feasibility of employing the speech reception threshold by bone conduction as a supplementary measure to the pure tone bone conduction test. The questions for study were, simply, the following:

1. What is the relationship by bone conduction between the average pure tone threshold for the frequency range 500 to 2000 cps and the speech reception threshold?

2. How does the relationship between speech and pure tones by bone conduction compare with its companion relationship by air conduction?

APPARATUS, CALIBRATION, AND PROCEDURE

Apparatus

The testing chamber at the Hearing Clinic of the University of Kansas Medical Center is similar to others in this country. It consists of a control room and a test room, with an observation window between the two. In the control room is located a Maico Console Research Audiometer. This assembly incorporates several channels which provide for the measurement of hearing by pure tones and by speech, either through the headphones or through a bone receiver. The equipment includes a playback, microphone, and VU meter so that speech tests can be performed using either recorded materials or monitored live voice. The latter procedure has, in general, been employed at the clinic and constitutes the method by which the data were obtained. Attenuation of pure tones is in the conventional 5 db.

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* References 1 to 3, 5, and 6.

† References 4, 8, 14, and 15.

‡ References 8, 13, and 15.

steps. The speech channel, however, is equipped with a set of Daven attenuators with a range of 110 db., thus permitting attenuation to 1 db.

The test room is a room within a room. It contains a Jensen speaker for free-field testing, a set of PDR-10 earphones mounted in MX-41/AR cushions, a Maico Model C bone oscillator, and a chair for the subject. The ambient noise level of this room, as measured with a General Radio Sound Surver Meter, Type 1555-A, is 35 db. on the A scale. On the C scale the level is 50 db. The room is, therefore, adequate for threshold measurements when earphones are utilized. As would be expected, a slight shift occurs in the threshold responses of normal hearing subjects by bone conduction.

Calibration

The pure tone generator, a Maico E-1 audiometer, of the assembly with earphones and bone receiver, is returned yearly to the factory for sound-pressure calibration. The E-1 audiometer, as well as the auxiliary channels for speech testing (monitored live voice and recorded speech) by air conduction, is periodically calibrated at the clinic by the method as advocated by Carhart.[4] The clinical calibration of pure tones by bone conduction is likewise accomplished by the Carhart method, and it has been applied to establish the threshold for speech by bone conduction.

Thresholds of hearing by bone conduction were measured at the mastoid bone with the contralateral ear masked by saw-tooth noise. The average masking effects of the noise on the threshold sensitivity of the contralateral ear by bone and air conduction had previously been determined, and these averages were used in setting the level of the noise when masking was needed. It has been a rule at the clinic to mask the contralateral ear during bone measurements when the difference between the two ears by air conduction exceeds 10 db.

Procedure

Speech reception thresholds by air conduction are measured monaurally in the hearing clinic. During the past year speech thresholds by bone conduction were included in the regular battery of tests which constitute an auditory assessment. The bone receiver was held in place against the mastoid by a steel headband. Care was taken to place the receiver against the area of maximum sensitivity. In the clinical procedure the speech reception test by bone conduction was administered immediately following the pure tone threshold by bone conduction. All pure tone thresholds were determined by the ascending method.

Records on 191 patients, ranging in age from 5 to 64 years, with a mean age of 34 years, were selected from the clinic files. These records yielded data on 353 ears which had been tested by pure tone and by speech air conduction audiometry. Two hundred seventy-one of the 353 ears had been given, in addition, bone conduction tests by pure tones and by speech. Data on known cases of brain damage, psychogenic deafness, severe nerve deafness, and malingering were excluded from the study. The data thus collected were subjected to statistical analysis.

Results

The analysis of the data is presented in the Table.

As shown in the Table the mean thresholds by air conduction for the pure tone average of frequencies 500 to 2000 cps and for speech reception were 40.76 and 41.60 db., respectively. The t-ratio (0.581) of the difference between the means is not statistically significant. Further, the F-ratio (1.05) indicates that the difference between the variances was not significant at the 5% level of confidence. The product-moment correlation of 0.92 between the two tests by air conduction indicates a high degree of relationship. It is higher than the results reported by Carhart.[1] This investigator obtained values for r ranging from 0.29 to 0.87, depending upon the pattern of pure tone hearing loss. The coefficient of 0.29 for marked high tone losses represents, however, an atypical result, the range for other classifications being from 0.71 to 0.87. The data of the present study, as previously observed, were selective. Re-

Analysis of Threshold Data on 353 Ears by Air Conduction and 271 Ears by Bone Conduction Determined by Pure Tone and Speech Audiometry

	Test	Mean (Db.)	Standard Deviation (Db.)	F-Ratio	t-Ratio	r
AC	P/T	40.76	18.94	1.05‡	0.581*	0.92
	S/R	41.60	19.40			
BC	P/T	19.04	14.00	1.00‡‡	0.002†	0.90
	S/R	19.05	13.97			

The meaning of the abbreviations is as follows: AC, air conduction; BC, bone conduction; P/T, pure tones; S/R, speech reception.

‡ = 0.05 P < (Therefore a nonsignificant difference between variances for air conduction).

‡‡ = 0.05 P < (Therefore a nonsignificant difference between variances for bone conduction).

* = 0.5 < P < 0.6 (Therefore a nonsignificant difference between the means for air conduction).

† = 0.9 < P (Therefore a nonsignificant difference between the means for bone conduction).

ferring to the Table, it is clear from the means and standard deviations by air and bone conduction that the population had predominantly conductive and mixed impairments. The ranges of hearing losses were, however, large.

The Table shows the mean hearing losses by bone conduction for pure tones and for speech to be almost identical, the former being 19.04 db., the latter 19.05 db. As one would expect, the *t*-ratio of the difference between the means is nonsignificant. The *F*-ratio between the variances of 1.00 is likewise nonsignificant. The coefficient of correlation by bone conduction between pure tones and speech was 0.90. It is higher than the correlations by air conduction as reported in the literature. While the likelihood of a bias in the data as a result of the method (monitored live voice) cannot be ignored, factors such as selectivity of the data plus the wide ranges of hearing losses by both bone and air conduction could tend to enhance the correlations. Another possible source of difference is that the present data resulted from monaural threshold measurements. In any event, one might expect a substantial correlation by bone conduction between speech and pure tones of approximately the magnitude of the relationship by air conduction, if the test-retest reliability of bone conduction by pure tones when compared with the test-retest reliability of air conduction by pure tones is used as an index. In other words, test-retest differences by pure tone bone conduction are only slightly poorer than test-retest differences by air conduction.[15] In addition, variability of normal thresholds by pure tone bone conduction has been shown to be only slightly greater than by air conduction.§

In reference to the questions which were posed at the beginning of this study, the following statements might be made:

First, the relationship by bone conduction between the speech reception threshold and the pure tone average of frequencies 500 to 2000 cps as expressed by the product-moment coefficient of correlation was found to be 0.90. This relationship is higher than had been previously expected on the basis of reported correlations between speech and pure tones by air conduction. While the presence of a bias in the data contingent upon the speech reception test method is possible, other factors, such as the selection of cases, the wide range of hearing losses, and the measurement of speech monaurally, could influence positively the size of the correlation.

Second, for these data the relationship by bone conduction between pure tones and speech was found to be only slightly poorer than the relationship between the two tests by air conduction. Further research concerning the problem will be conducted employing standardized test records.

COMMENT

Speech testing by bone conduction has been found to be a useful adjunct to the regular battery of tests which comprise an auditory assessment at the University of Kansas Medical Center. Aside from its use as a check upon the reliability of the pure tone bone conduction threshold there are several other advantages. First, the test has been particularly useful in contributing to a diagnosis of conductive impairment, especially with small children, when it has not been possible to obtain reliable information by pure tones. In such instances the air-bone gap by speech audiometry has proved to be very helpful. In conjunction with the speech reception tests discrimination lists are frequently employed to afford a more detailed analysis. For example, neural involvement has been detected (and later confirmed by pure tone tests) in children by virtue of the discrepancy in PBK scores by bone conduction between the norm established here at the clinic and the obtained score. Another comparison which has provided useful information is between the PB score by bone conduction relative to the patient's speech reception threshold by bone conduction and

§ References 8, 9, 14, and 15.

the PB score by air conduction with reference to the patient's speech reception threshold by air conduction.‖

Second, the speech reception threshold by bone conduction has been utilized to supplement the pure tone bone conduction finding in the prediction of suitable cases for fenestration surgery. In the procedure the average of the Carhart notch at frequencies 500, 1000, and 2000 cps (The Carhart notch is a mechanically induced depression in the bone conduction curve which is not attributable to neural degeneration) or 10 db. is subtracted from the obtained speech reception threshold by bone conduction. This result plus 25 db. is taken as the best estimate of the postoperative hearing level.

CONCLUSIONS

1. Speech reception tests by bone conduction correlate well with the average pure tone threshold by bone conduction for frequencies 500, 1000, and 2000 cps.

2. Clinical advantages of speech testing by bone conduction include the following: (*a*) It provides a check on the reliability of pure tone bone conduction. (*b*) It is an aid in prediction of the result of the operative procedures for otosclerosis, where accuracy in determination of the cochlear involvement is paramount.

‖ PB tests by monitored live voice are given 25 db. above the subject's speech reception threshold. At this level a score of 90%-100% is obtained by persons with normal hearing as well as by persons who have conductive impairments. Persons with neural impairment exhibit varying degrees of discrimination difficulty.

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Case Reports

THE SURGICAL APPROACH TO BRAIN ABSCESS OF OTITIC ORIGIN

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THE INTRACRANIAL complications of mastoiditis have become a comparative rarity since the advent of the antimicrobials. Those of us who have seen a large ward in the Allegemeine Krankenhaus filled with patients with these complications, including several brain abscess cases, can testify to it. This change was well developed in a symposium on the subject at the Western Section of the American Laryngological, Rhinological and Otological Society, Inc., in January, 1955, and was discussed by a panel of neurologists, neurosurgeons, and otologists. They failed, however, to touch on what has long been a moot question—the proper surgical approach to brain abscess of otitic origin.

It is well known that the proportion of brain abscesses secondary to mastoiditis is very high. Stuart and associates [1] in an excellent review of 125 cases report a large proportion to be found as complications of chronic middle-ear infections as against acute ones (3% of 1400 chronic cases to 0.5% of acute cases). Of 61 cases of otitic origin 41 were cerebral and 20 cerebellar. Pertinent to the purpose of this paper is the following statement made by Stuart and associates: "It [the abscess] was almost always located within that part of the brain contiguous with the site of primary infection."

In the language of the labor unions, we have long been faced with a friendly jurisdictional dispute. Should a brain abscess be drained by the otologist through the infected area from which it sprang or by the neurosurgeon through a more remote and probably uncontaminated field? The argument

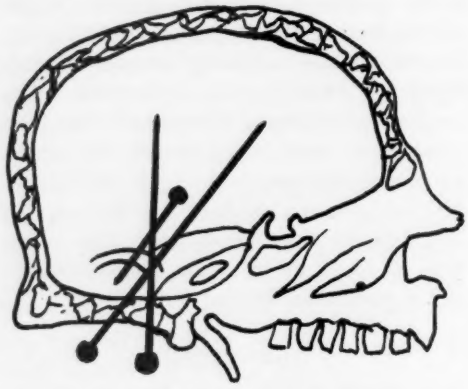

Fig. 1.—Aspirating needle radiating from mastoid area. (Reproduced with permission: Cunningham Textbook of Anatomy, Oxford University Press, Inc.)

in favor of the former is that drainage through the dura of the tegmen antri and tympani or through the posterior fossa serves as a more direct approach. The otologist is in a position to uncover an epidural abscess, expose infection of the brain membranes, and explore for a fistulous tract to the abscess, if such a tract exists. If there is no obvious fistula, he can pass the brain needle in any radiating direction from the infected nidus (Fig. 1). The argument against such an approach has been that micro-organisms would be carried by the aspirating needle through the infected area into uninfected brain tissue. It would seem that this objection can be overcome with the use of antibiotics and some such technique as is used in the following case report, wherein I should like to defend the otologic approach. Coates * and others feel as I do in this matter.

REPORT OF A CASE

A girl, aged 8 years, with an acute exacerbation of a left chronic mastoiditis was seen on Feb. 20, 1954, by residents at the outpatient department of the Sonoma County Hospital. She was given anti-

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* Coates, personal communication to the author.

Fig. 2.—Diagram of frontal section through the brain at mastoid level showing avenue of approach to abscess in case reported. (Reproduced with permission: Cunningham Textbook of Anatomy, Oxford University Press, Inc.)

biotics and pain medication and sent home. On March 1 she was admitted as a bed patient with symptoms of an intracranial complication. A neurosurgeon called in consultation made a presumptive diagnosis of temporal lobe abscess. The diagnosis became more positive after a ventriculogram, which showed displacement of the ventricles to the right.

The neurosurgeon attempted aspiration of the abscess through two burr holes in the parietal and temporal regions, without success. The child became completely comatose with signs of increasing intracranial pressure. I was asked to see the child and advised immediate exploration of the mastoid, dura, and brain through that area. On March 5 through a postauricular incision an infected mastoid was exenterated, with exposure of the dura over the tegman antri and tympani. There was no gross evidence of infection of the membranes. A circle of dura, 1 cm. in diameter, was electrocoagulated to produce an inflammatory dam against extension of infection from within the circle. Iodine tincture was applied, and a small crucial incision was made in the center of the dural circle. No pus was encountered. An aspirating brain needle was introduced in a medial and superior direction, where at a depth of 2.5 cm. a rubber-ball resistance area was encountered (Fig. 2). A quick short thrust penetrated what proved to be the abscess wall, and more than 30 cc. of creamy staphylococcic pus was evacuated under pressure. A small polyethylene tube was introduced into the cavity and allowed to remain 24 hours.

The course of the case proved that the abscess was fortunately not multiple. The child's recovery was rapid and uneventful, with no obvious defects except two permanent pulsating cranial defects and an unhealed mastoid cavity. Six weeks later the radical mastoidectomy was completed. At the time of publication, nearly two years later, no sequelae have developed and the child is normal.

It is not my purpose to attempt to exclude the valued advice or services of our conferees who are skilled in the care of diseased brain states. Team work is needed. It might occasionally be desirable to treat the abscess by some other method, such as marsupialization with excision of the abscess wall.† There are those who feel that postoperative scar-tissue contraction with consequent seizures would be reduced thereby. One might reason that this treatment should be reserved for those cases which did not do well with simple aspiration. If much tissue damage was produced by this more major procedure, a larger infected area would be productive of more gliosis and fibrosis, with still greater contraction.

The otologist need not feel at a disadvantage in the search for a brain abscess of otitic origin. Automatically he is in a position to approach the infected mass in a manner which should allow a substantial degree of success, and with it the simultaneous exposure and evaluation of all the infected area from which the infection sprang. The above-mentioned precautionary measures in walling off and sterilizing the dura, plus the added protection now offered by the antibacterial agents, give the otologist a positive advantage in the treatment of this complication.

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† Morrison, L. F., personal communication to the author.

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MALIGNANT SCHWANNOMA OF TIP OF NOSE

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MALIGNANT schwannoma occurring on the tip of the nose is an extremely rare condition. The fact that it occurred in a person who had irradiation to the face for eczema 15 years prior to this episode adds further interest.

Benign and malignant neurilemmoma can occur in any peripheral nerve in the body possessing a neurilemma. The malignant schwannoma can occur in any age group but is most frequently seen after the fifth decade. No specific causative factors are known. There is, however, a definite relationship with disseminated neurofibromatosis. Approximately 50% of the malignant neurilemmomas reported have occurred in persons suffering from disseminated neurofibromatosis (von Recklinghausen's disease). There has been no proved instance of association with benign neurilemmoma and no record of a benign neurilemmoma ever having turned into a malignant neurilemmoma.

When the disease is associated with disseminated neurofibromatosis, the prognosis is very poor, with aggressive and extensive infiltration into the surrounding tissue and early blood-stream spread to the chest, brain, liver, and bone. Lymphatic invasion is not common. There is no evidence of limitation by capsular formation. The nerve in which it arises is destroyed, and the tumor enlarges underneath the skin, forming a semifixed mass which eventually ulcerates through the skin. Microscopically, the Schwann cells tend to grow in cords and palisades separated by delicate reticular fibers.

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The prognosis in a solitary malignant schwannoma not associated with von Recklinghausen's disease is good. The tumor is not radiosensitive and is best handled by wide surgical excision.

REPORT OF CASE

A woman aged 30 related that at approximately age 15 she developed severe eczema of her face and body, and was treated intermittently with x-radiation over a period of three years. The number of exposures to x-ray were calculated to be approximately 150. The allergic dermatitis was complicated by asthma and allergic rhinitis. There was considerable improvement in the dermatitis of the face following this x-ray therapy. Approximately seven years after the x-ray treatment, the patient noted that the skin had atrophied and had a tendency to break down. This was particularly marked about the upper lip and nose.

Examination in 1947 revealed thin, atrophic, telangiectatic skin covering the brow, cheeks, nose, and upper and lower lips. This condition existed to a minimal degree on the neck. There was a chronic ulcerating fissure in the left nasolabial fold.

Since 1949, the skin of the nose and upper lip had manifested multiple degenerative effects. Three postirradiation ulcers were removed from the nose and one fibrosarcoma from the right upper lip. In September, 1953, the patient developed a slow-growing, well-fixed, subcutaneous mass on the tip of the nose on the left side. It eventually ulcerated through the skin. The mass was painless. As a

Fig. 1.—Malignant schwannoma involving the tip of the nose. Severe x-ray dermatitis of face is evident.

Fig. 2.—Postoperative view of composite graft (2 by 2 cm.) replacing left ala, tip and portion of columella and right side of nose.

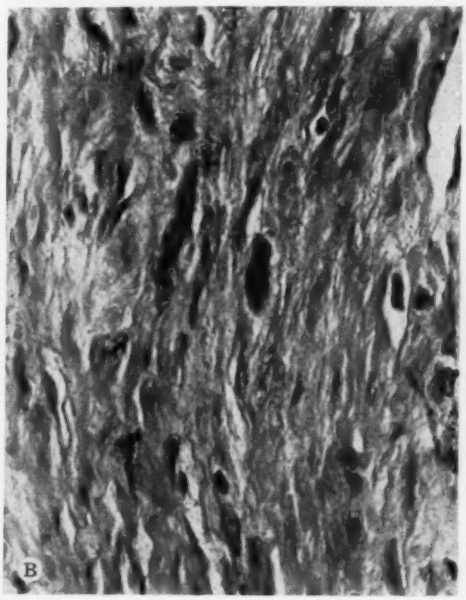
matter of fact, the earliest symptom was a loss of sensation on the tip of the nose.

The entire tip of the nose was widely excised in a three-dimensional manner. The excision included part of the left ala, a portion of the columella, the entire tip, and a portion of the right side of the nose. It was a through-and-through excision, including the skin lining the vestibule. The wound was repaired immediately by the use of a 2 by 2 cm. composite graft from the region of the right ear lobe. Although the graft was taken according to pattern, it was necessary to adjust it on the nose because the defect not only included the tip and ala of the nose but also the columella. The donor area in the ear was corrected by a postauricular flap. The composite graft in the nose had a 100% "take."

Fig. 3.—Donor site on ear, healed.

Fig. 4*A*.—The neurilemmal cells are plump but remain spindle-shaped. The fasciculation, which helps differentiate this tumor from one of fibroblastic origin, is still prominent. The nuclei show hyperchromatism and considerable variations in size. Mitoses are rare. Nuclear detail is not too evident.

Fig. 4*B*.—A higher magnification of the area in Figure 4, showing fasciculation, the fibers and the cell nuclei with their irregular distribution of the chromatin, hyperchromatism, and other changes indicative of malignancy.



COMMENT

It is unusual to obtain a perfect take of such a large composite graft (2 by 2 cm.) in a postirradiated, devitalized tissue bed. The principle of cooling (mild refrigeration) has been applied to large grafts which have been transplanted to devitalized recipient sites. This reduced temperature diminishes the biologic demands of the transplant while it is establishing its connection with the host. When the metabolic velocity is diminished, the vascular connections of the graft have a greater interval of time in which to accommodate themselves to the tenuous support of a poor tissue bed.

SUMMARY

An unusual case of malignant schwannoma of the tip of the nose is presented. It was treated by a wide surgical excision with immediate reconstruction by a large composite graft. A special cooling technique was employed to the graft to enhance the "take."

News and Comment

ANNOUNCEMENTS

A. M. A. Annual Meeting, Chicago, June 11-15, 1956.—The 105th Annual Meeting of the American Medical Association will be held in Chicago, June 11-15, 1956, with a full program of lectures, television, motion pictures, and scientific exhibits.

The lecture program for each Section is prepared by the Section Secretary, and prospective participants should communicate as soon as possible with the Secretary of the Section before which they hope to read a paper. Application blanks for space in the Scientific Exhibit are now available and may be obtained from the Section Representative to the Scientific Exhibit.

Section on Laryngology, Otology and Rhinology

Secretary: Hugh A. Kuhn, M.D., 112 Rimbach St., Hammond, Ind.

Exhibit Representative: Walter E. Heck, M.D., Stanford University Hospitals, San Francisco 15.

Additional information may be obtained from the Secretary, Council on Scientific Assembly, American Medical Association, 535 N. Dearborn St., Chicago 10.

AN UNUSUAL FOREIGN BODY, A MARBLE, IN THE ESOPHAGUS

JOHN R. AUSBAND, M.D.
JAMES A. HARRILL, M.D.
and
HOOPER D. JOHNSON, M.D., Winston-Salem, N. C.

FOREIGN bodies of practically every description have been found in the esophagus. Many present problems in removal due to the material of which they are composed, position, location, and shape. Jackson [1] reported the removal of a marble from the esophagus, just below the cricopharyngeus, of a 4-month-old child. The forceps used was the safety pin closer used as a mechanical spoon. Jackson and Jackson [2] describe a method for removing a marble from the bronchus, but this method was not applicable in these cases, since the proper-sized forceps was not available. The following case reports and the handling of the problem are, we believe, self-explanatory.

REPORT OF CASES

CASE 1.—A 3-year-old white girl was admitted to the North Carolina Baptist Hospital on Aug. 14, 1954. The history, as obtained from the father, was that the child was on the floor at home playing with marbles when she began to "cough and choke." There was no known cyanosis and no vomiting. The "coughing and choking" continued, and the child was taken to her local physician, who had roentgenograms made. A diagnosis of esophageal foreign body was made, and the child was referred to this hospital for treatment.

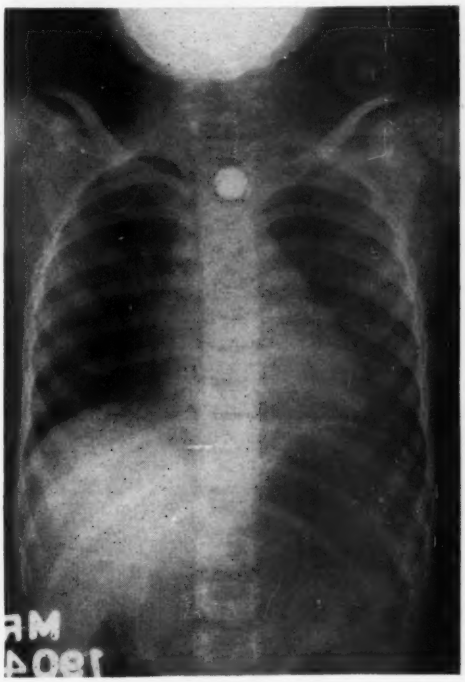
Physical examination showed temperature to be 100.2 F (rectally), pulse rate 110, respiration rate 28, and blood pressure 110/70. The child was well developed and well nourished, showing no cyanosis and in no distress. No abnormalities were noted in the examination of the head, eyes, ears, nose, and mouth. The lung fields were resonant to percussion, and normal breath sounds were heard by auscultation. There were no areas of altered breath sounds. Abdominal organs were not enlarged, and the extremities were normal.

Accessory clinical findings included urine amber in color, specific gravity of 1.035, pH of 7.0, no albumin or sugar, and microscopically a few phosphate crystals were seen. The blood contained 11.0 gm. of hemoglobin, 5,000,000 red blood cells per cubic millimeter, and 12,000 white blood cells per cubic millimeter. Differential count showed 63% segmented PMN, 1% nonsegmented PMN, no eosinophiles or basophiles, 30% lymphocytes, and 6% monocytes.

Chest x-ray on admission showed a rounded foreign body in the esophagus at the level of the cricopharyngeus (Fig. 1). The trachea was deviated toward the right.

Course in hospital: The evening of admission the child was taken to the endoscopic room, at which time the foreign body could be palpated with

Fig. 1 (Case 1).—Foreign body in esophagus at level of the cricopharyngeus. The trachea is deviated to the right.



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From the Department of Otolaryngology, Bowman Gray School of Medicine of Wake Forest College.

the tip of the finger. It was exposed with the child-size esophagoscope and noted to be a blue marble, but no instrument would grasp it. However, it was felt that the marble was pushed below the cricopharyngeus. Check films the next day showed that it was lodged at the level of the arch of the aorta. Again, esophagoscopy was carried out, but an instrument which would grasp this perfectly round, smooth marble was not at hand. The procedure was discontinued. The following day the use of the Johnson stone basket, No. 9 F., was suggested, and it was found that a marble of similar

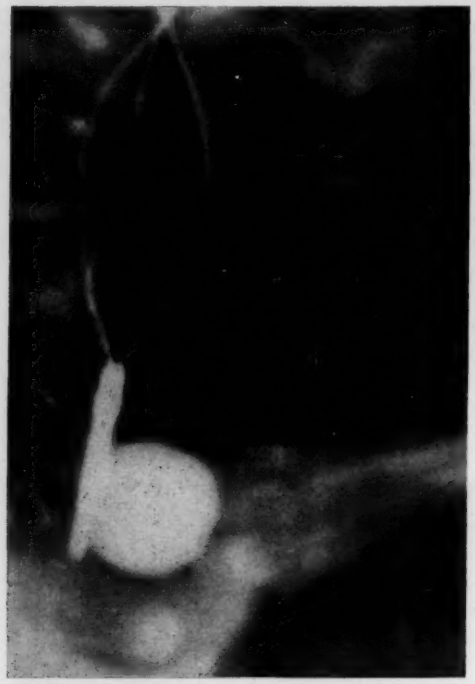


Fig. 2.—The stone basket is in the esophagus, and the tip is in relation with the marble.

size would enter the basket without apparent difficulty. Accordingly, the child was taken to the x-ray suite, and, under fluoroscopic guidance, the stone basket was introduced into the esophagus (Fig. 2). The tip passed the marble, and the marble promptly entered the basket (Fig. 3). The basket was very carefully removed, bringing the foreign body with it. There were no apparent complications, and the following day x-ray examination of the esophagus was carried out. This showed no abnormality, and the child was discharged on Aug. 20, 1954, completely asymptomatic.

Figure 4 shows the marble and stone basket with a scale for comparative size.

Case 2.—A 2-year-old white boy was admitted to the hospital on April 10, 1955. The child was

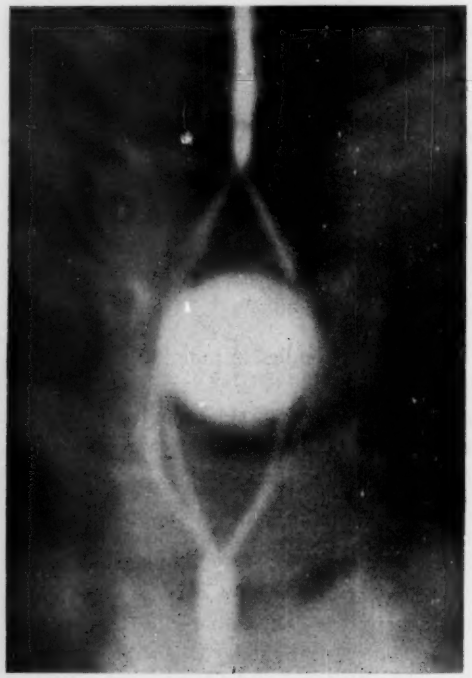
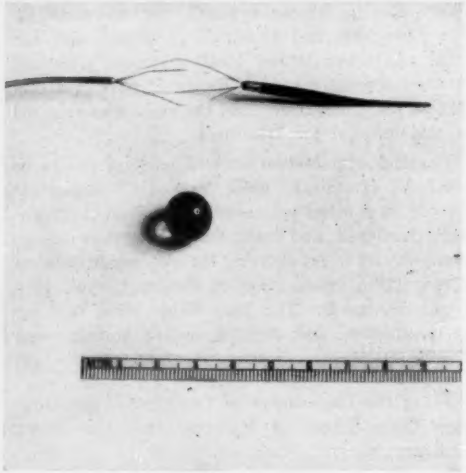


Fig. 3 (Case 1).—The marble has entered the stone basket and is ready for removal.

well until five days before admission, when he began regurgitating everything taken by mouth. X-ray studies, including barium studies, at a hospital in a neighboring city, revealed a "marble in the mid-esophagus." The parents could not recall an episode of choking or vomiting but did recall that the child was playing with a set of

Fig. 4.—The Johnson stone basket and the marble removed in Case 1.



"Chinese checkers," in which small glass marbles are used, on or about the day of the onset of regurgitation. The child was therefore transferred to this hospital for treatment.

Physical examination: Pulse rate 88, temperature 99.2 F (rectally), respiration rate 18. The patient was a well-developed and well-nourished white boy in no distress and well hydrated. There were no physical abnormalities of the ears, nose or throat. Pupils were round, regular, and equal and reacted to light and accommodation. The heart was not enlarged. Rate and rhythm were regular; no murmurs were heard. The lung fields were clear

Fig. 5 (Case 2).—Lateral film showing marble in the esophagus, below the arch of the aorta.

to percussion and auscultation. The trachea was in the midline. The abdomen was soft, nontender, and no masses or organs were palpated. Reflexes were physiologically active throughout.

Accessory clinical findings: Blood—11.3 gm. of hemoglobin; 3,840,000 red blood cells per cubic millimeter; 12,000 white cells per cubic millimeter; differential count showed 53% segmented PMN, 4% nonsegmented PMN, 4% eosinophiles, 34% lymphocytes, and 5% monocytes. Urine was clear and yellow, specific gravity being 1.024, reaction neutral, negative for albumin and sugar. Microscopically there was a rare granular and epithelial cast and 1 to 3 white blood cells per high-power field.

X-ray films on admission (Fig. 5) showed a rounded opaque foreign body lying in the esophagus, just inferior to the bifurcation of the trachea.

On April 12, 1955, the child was taken to the x-ray suite. Under fluoroscopic guidance the Johnson stone basket was introduced into the esophagus. The marble was located in a larger, looser segment of the esophagus than in the previous case and was caught in the basket with somewhat more difficulty than had been encountered at that time. It was, however, caught in the basket and brought out without any further difficulty.

After removal of the marble, a barium study of the esophagus was done and this demonstrated no disease of the esophagus.

The child was discharged on April 13, 1955, entirely well.

COMMENT

We were informed by the members of the department of urology that on several occasions one of the wires of the Johnson stone basket has broken, with the serious complication of perforation of the ureter. They are now using a similar basket, the flexible leaves of which are made of nylon. We have ordered a basket of this type, of larger size than usually required to remove a ureteral stone, and it is now being constructed.

SUMMARY

A 3-year-old child and a 2-year-old child with a marble in the upper and lower portions of the esophagus respectively were seen. The first marble could not be removed endoscopically, since a forceps of proper size was not available, but was successfully removed with the Johnson stone basket. A similar foreign body was seen in the second child, and this was removed in a similar fashion, but somewhat more difficulty was encountered due to the looseness of the esophagus in its lower portion. The wire-type Johnson stone basket is being discarded in favor of a nylon basket.

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METASTATIC HYPERNEPHROMA OCCURRING IN NASAL SEPTUM

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PRIMARY malignant tumors in the nasal cavity, although rare, have been recognized for a long time. These tumors usually occur as polypoid masses arising from the mucosa of the nose or the nasal sinuses. In the latter case, symptoms of bleeding and pain may be noted or presence of the tumor may first be recognized by radiological manifestations of bone destruction. In many instances, carcinoma may be found on routine pathological examination of material removed during radical procedures on the antrum. By far the greater number of cases are squamous-cell carcinomas, although a small percentage are adenocarcinomas, of which the mucinous type is distinctive.

McComb and Martin [1] published a report in 1942 analyzing 65 cases of cancer of the nasal cavity. Of these, 11 cases were adenocarcinomas (17%). In a thorough search of the literature, both textbooks and medical journals, I was unable to find any reference to metastasizing tumors in the nasal cavity. While the condition is probably rare, one would still expect to find some cases if efforts were made to look for them.

Rusche [2] has pointed out that hypernephromas have the singular capacity of giving rise to solitary metastasis, often many years after the removal of the primary tumor. This author also points out that metastasis of hypernephroma may take place in practically any organ in the body. It is also believed that where metastasis is solitary removal of both the primary and the solitary metastatic lesion will effect a cure.

Barney and Churchill in 1939 claimed that the thorax and cranial contents are the most likely sites for metastatic adenocarcinoma of the kidney. Johnson and Straus [3] have reported a case of hypernephroma metastasizing in the small intestine, causing an intussusception. This is one of the two cases reported in the last 25 years. Stewart and Bruce [4] recorded a case of a hypernephroma that metastasized to the mandible. There were also cases reported of metastases in the vagina,[5] pancreas,[6] lungs,[7] mediastinum,[7] and thyroid.[8] All these cases prove only the fact that no organ in the body is exempt from the metastasis of a hypernephroma.

While metastasis via the lymph channels is by far the commonest method of spread of a carcinoma, blood-stream metastasis is also important. The most usual site of blood-borne metastasis is the lungs, since all blood from the systemic circulation passes through the pulmonary capillaries.

McDonald and Priestley [8] have dissected 509 kidneys removed surgically because of hypernephroma and established tumor thrombosis of the renal vein or neoplastic involvement of lymphatics in about 54% of cases. Thus, embolism from the renal vein is one of the important channels of spread of tumor cells.

There is no unanimity concerning the origin of this peculiar tumor known as hypernephroma. This name was given by Grawitz when he published a report on these tumors in 1883. He produced evidence that they arose from embryonal rests of adrenal cortical tissue. These rests sometimes are encountered beneath the capsule of the kidney normally. Also, the microscopic appearance of the tumor resembles

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that of adrenal cortex, the tumor being composed of clear cells showing hydropic changes or containing abundant doubly refractile cholesterol esters; the cells may be arranged in cords as in adrenal cortex. The evidence against the above theory is that these tumors never give rise to endocrine or sexual disturbances.

The symptoms of any nasal tumor, whether benign or malignant, are (1) nasal obstruction, (2) nasal discharge, and (3) epistaxis.

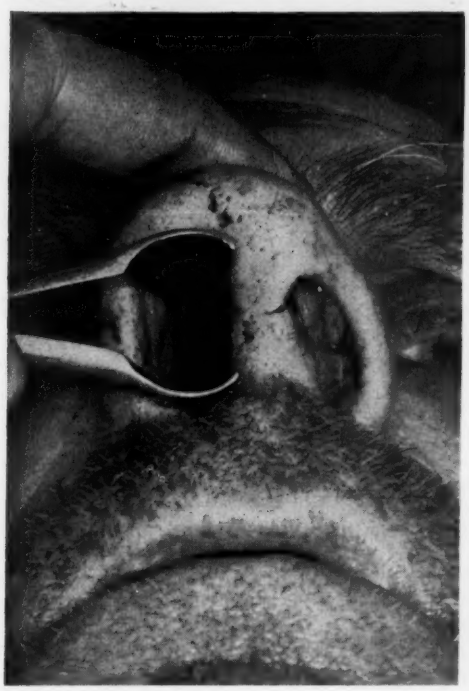


Fig. 1.—The right nasal fossa is held open with a nasal speculum. The polypoid mass is seen hanging from the septal wall.

While the first two symptoms are frequent in the commonly encountered nasal polyps which are benign, epistaxis is an unusual symptom in uncomplicated cases. It is very essential to investigate the underlying cause of nose bleeding in the case of nasal polyps, as nasal polyps may represent an end-result of obstruction and edema caused by some other pathology.

The case here presented is significant in that the diagnosis of hypernephroma was

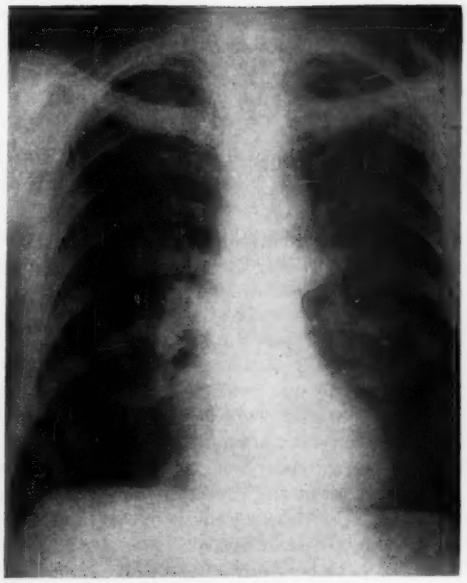


Fig. 2.—A posteroanterior view of the chest shows a mottled area of increased density in the lower portion of the left hilar area extending into the left lower lung field. Rounded and fairly discrete areas of increased density are also scattered through the lung field. These probably indicate metastatic disease (picture taken on Sept. 9, 1954).

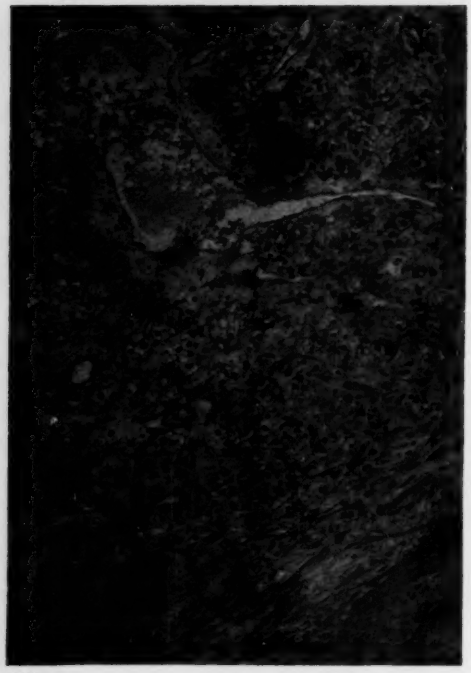
Fig. 3.—Chest film taken on Nov. 15, 1954, as a follow-up study. Here are seen many fairly well-defined areas of increased density scattered throughout both lung fields. These probably represent extensive metastatic foci.

made from biopsy material from the nose before it was known that the patient had a renal tumor.

REPORT OF CASE

A 62-year-old white man was admitted to St. Luke's Hospital Ear, Nose, and Throat Service on Sept. 9, 1954, with a history of bleeding from the right nasal fossa. Patient gave a history of recurrent nosebleeds for about one and a half years prior to this admission. These were controlled each time by simple measures administered by his private physician. Patient also gave a history of admission to another hospital for a severe nosebleed about a year previously. This was treated by local measures, and the patient was discharged home as cured. The nose bleeding always came from the right side. There was no history of hypertension or blood dyscrasia. Patient also gave a history of admission to St. Francis' Hospital, Bronx, on April 7, 1954, for pain in the right kidney region. He had had a nephrectomy on April 8. The pathological diagnosis was hypernephroma.

Fig. 4.—The section is composed of clear cells with uniform-sized hyperchromatic nuclei in an alveolar arrangement. The delicate fibrous stroma is highly vascular. Fibrin, hemorrhage, and edematous, proliferating, fibroblastic tissue cover the surface. The latter is heavily infiltrated by polymorphonuclear cells, lymphocytes, and macrophages.



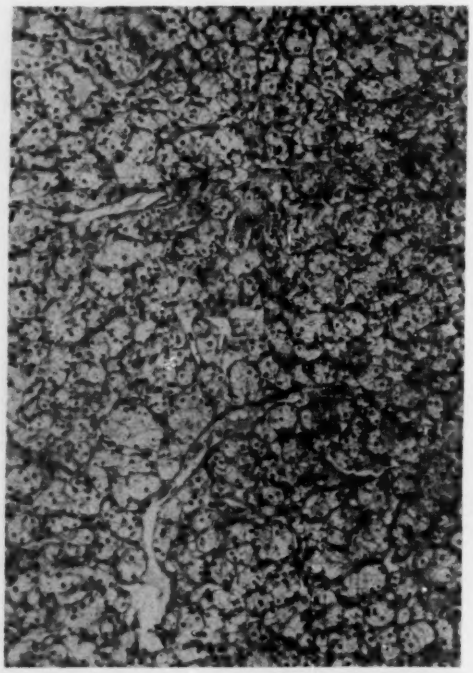


Fig. 5.—The same section as in Figure 4 under higher magnification. The typical alveolar pattern of the hypernephroma is seen.

The present incident of bleeding started spontaneously on Sept. 8, 1954, and the private physician was unable to control it with anterior packing. Patient was admitted to the ward, and immediately a ½ in. petrolatum gauze pack was tightly applied in the right nasal fossa; the bleeding was controlled. The packing was removed on Sept. 10, but the bleeding recurred so that the packing had to be reapplied. The bleeding was completely controlled by Sept. 13 (fifth day after admission).

A complete E. N. T. examination was then done. A polypoid mass was seen coming down from the nasal septum at about the level of the middle turbinate (Fig. 1). It was red and mobile. Any attempt to manipulate it started fresh bleeding. The airway on the right was completely blocked by the presence of the polypoid mass. The tumor was not visualized on the postnasal mirror examination. Posterior choanae were free. The investigations included radiological examination of the nasal sinuses and chest. The sinus x-rays were reported as negative. The chest examination revealed a small dense shadow in the left hilar region, suggestive of malignancy of the lung (Fig. 2).

On Sept. 13, small pieces of biopsy material were taken from the polyp and were sent for pathological examination. Patient had bleeding after this, and the nose was packed with petrolatum packing. On Sept.

16, attempts at removal of the packing started bleeding again and the nose was repacked. The pathology report was as follows: "Tumor consists of loosely packed fibrous stroma showing edema with portions of a respiratory epithelium covering. Hemorrhage and necrosis are present in some portions. An area of granulation tissue is present. Within granulation tissue there are round nests of clear cells with uniform nuclei in alveolar arrangement. In many cells, the cytoplasm is vacuolated. These cells probably represent a metastatic clear-cell carcinoma.

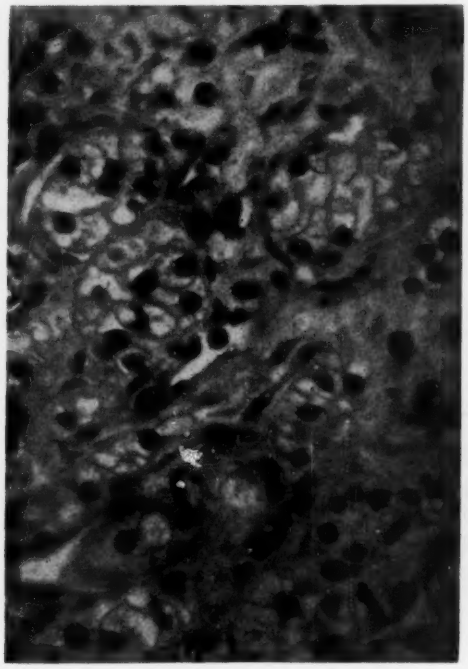


Fig. 6.—The section of hypernephroma shown in Figures 4 and 5 under the highest power.

"Diagnosis: Nasal polyp, probably containing metastatic adenocarcinoma of the kidney."

As the appearances on the pathological section were not conclusive, another biopsy specimen was taken from the tumor on Sept. 23. The pathology report stated: "The bulk of the section is composed of clear cells with uniform-sized hyperchromatic nuclei in an alveolar arrangement. The delicate fibrous stroma is highly vascular. Fibrin, hemorrhage, and edematous, proliferating, fibroblastic tissue cover the surface. The latter is heavily infiltrated by polymorphonuclear cells, lymphocytes, and macrophages and in areas is necrotic. On the surface, in a few areas, are short strips of one to two layers of epithelial cells.

"Diagnosis: Nasal polyp, containing metastatic adenocarcinoma of the kidney (hypernephroma)" (Figs. 4, 5, and 6).

Treatment and Course.—An operation to remove the tumor was not contemplated for two reasons: 1. The lesion was adherent to the nasal septum, and its complete removal would require a mutilating operation. 2. There was a lesion in the hilar region of the lung which was most probably another metastasis. Thus, local operation was unlikely to cure the patient. Hence, the treatment was directed to measures to control recurrence of nose bleeding. The patient was referred to the Radiotherapy Department for consultation. The following opinion was given: "Chest film shows multiple bilateral varying-sized metastatic lesions. Therapy, therefore, becomes purely palliative. This lesion in the nose is a most unusual one and being a hypernephroma may or may not respond to radiation, since their behavior varies widely. We should, however, be able to control bleeding, and, with that as our primary objective, we will start the therapy."

Therapy: Between Sept. 30 and Oct. 20, 1954, radiation therapy was administered to the lesion locally through an anterior nasal portal. Therapy was given daily five times a week. A total of 15 treatments was given. Each treatment delivered 200 r in air, thereby making a total dose of 3000 r. The factors used were 200 kv., 20 ma., Thoraeus filter, half-value Layer (hvl) of 4 mm. copper, 50 cm. T. S. D., and a 6×6 cm. portal.

After the institution of radiation therapy, the patient did not have any further episodes of bleeding from the nose. By the time the radiation therapy was completed, the tumor mass had regressed to less than half its original size. It was covered with white slough. Patient, however, was generally weaker and paler and had to be treated with blood transfusions. To study the cell changes in the tumor, another biopsy specimen was taken on Oct. 26, six days after completion of the radiation therapy.

The pathology report was as follows: "The surface is covered by a rather thick layer of fibrin, polymorphonuclear cells, and round cells which are undergoing necrosis and degeneration. Beneath these are sheets of large polygonal cells with clear cytoplasm and irregular nuclei which take a deep stain. Many small blood vessels are seen in this region, and a few scattered hemorrhages are noted. Other areas are undergoing necrosis.

"Diagnosis: Nasal polyp containing metastatic adenocarcinoma of the kidney (hypernephroma) undergoing necrosis."

Subsequent Course: Patient's clinical course subsequently was rapidly downhill, with cachexia, secondary anemia, and enlargement of the liver. Further treatment included opiates for pain and discomfort in the abdomen and infusions for correction of fluid and electrolyte balance. Patient went into

coma and died on Nov. 25, 1954. A postmortem examination was done, and in addition to the metastasis in the nose, metastatic carcinoma was found in the lungs, liver, thyroid, peritoneum, and lymph nodes of the abdomen. Changes of amyloidosis were found in spleen and kidneys.

SUMMARY

Primary carcinomas in the nasal cavity are rare, and secondary carcinomas in this area are still rarer. Metastatic carcinomas in the nose are most probably blood-borne.

A nasal polyp causing epistaxis must be viewed as suggestive of underlying malignancy.

A case is reported in which nasal metastases of hypernephroma caused recurrent episodes of epistaxis.

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News and Comment

ANNOUNCEMENTS

Postgraduate Courses in Bronchoesophagology and in Laryngology and Laryngeal Surgery.—The following courses are to be given in the Department of Laryngology and Broncho-Esophagology, Temple University Hospital and School of Medicine:

Postgraduate Course in Bronchoesophagology
Feb. 6th-17th, 1956
May 28th-June 8th, 1956
Sept. 17th-28th, 1956

Postgraduate Course in Laryngology and Laryngeal Surgery
April 23rd-May 4th, 1956
Nov. 5th-16th, 1956

These courses are all to be given under the direction of Dr. Chevalier L. Jackson and Dr. Charles M. Norris. The tuition fee for each course is $250.00.

Further information and application blanks can be obtained from Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.

Special Reports

HEARING REHABILITATION CENTERS IN THE UNITED STATES

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AND
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Glenview, Ill.

THE FOLLOWING listing of hearing centers in the United States was compiled from questionnaires sent to the centers and returned during the period of Nov. 1, 1954, to March 1, 1955.

When questionnaires were not returned, the information from the previous survey [1] was used. A blank space was left for any newly listed centers which did not return a questionnaire. Twelve new centers were added in this year's listing, including all the Veteran Administration audiology centers.

The following is the key to abbreviations in the list and Table.*

Medical Supervision
- Oto. Otologist
- C Consultant otologist
- R Resident otologist
- Phys. Physician
- C Consultant physician
- R Resident physician

Referrals
- Med. Referrals from doctors
- Ex. Exclusive medical referrals
- P Partial medical referrals
- O Other referrals from nonmedical sources

Restrictions (on clientele accepted)
- V Restricted to veterans only
- VP Veterans and general public
- P General public
- H Hospital patients only
- C Children only

Personnel (certification in American Speech and Hearing Association)
- M Member
- F Fellow
- AH Advanced Hearing Certification
- AHP Application pending for Advanced Hearing Certification
- BH Basic Hearing Certification
- BHP Application pending for Basic Hearing Certification
- AS Advanced Speech Certification
- ASP Application pending for Advanced Speech Certification
- BS Basic Speech Certification
- BSP Application pending for Basic Speech Certification

Equipment
- Pure-Tone Aud. Pure-tone audiometers
- Speech Aud. Speech audiometers
- Sound Rooms
- S Single sound room
- D Double sound room set-up
- PGSR Psychogalvanometer
- Special Other equipment such as audio-delay unit, etc.

Services
- Pure-Tone Audio. Pure-tone audiometry
- Speech Audio. Speech audiometry
- H/A Selection Testing and fitting of hearing aids for the patient
- H/A Demon. Trial of hearing aids without testing
- Lipreading Classes in lipreading training
- Auditory Train. Classes in auditory training
- Speech Train. Speech improvement therapy
- Psych. Testing Psychometric testing
- Counsel. and Guid. Counseling and guidance in problems of hearing loss and parental counseling

Accepted for publication May 17, 1955.

From the University of Denver (Mrs. Downs). Executive Secretary of the Audiology Foundation (Mrs. Carson).

* Note: X indicates the item or service is offered.

Number of Center	Medical Supervision: Oto. C	Medical Supervision: Oto. R	Medical Supervision: Phys. C	Medical Supervision: Phys. R	Referrals: Med. Ex.	Referrals: Med. P	Referrals: O	Restrictions	Personnel: Total Number	Degrees: B.A.	Degrees: B.S.	Degrees: M.A.	Degrees: M.S.	Degrees: Ph.D.	Certification: M	Certification: F	Audiology: AH	Audiology: AHP	Audiology: BH	Audiology: BHP	Speech: AS	Speech: ASP	Speech: BS	Speech: BSP	Equipment: Pure-Tone Aud.	Equipment: Speech Aud.	Equipment: Sound Rooms: S	Equipment: Sound Rooms: D	Equipment: PGSR	Equipment: Special	Services: Pure-Tone Audio.	Services: Speech Audio.	Services: H/A Selection	Services: H/A Demon.	Services: Lipreading	Services: Auditory Train.	Services: Speech Train.	Services: Psych. Testing	Services: Counsel. and Guid.
1	x	..	..	x	..	x	..	P	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	1	..	..	..	x	x	x	x	x	x	x	x	x
2	x	x	x	..	..	x	x	VP	4	..	..	2	..	2	4	2	1	..	..	..	1	..	1	..	2	.	2	1	..	..	x	..	x	x	x	x	x	x	x
3	..	..	..	..	..	x	x	VP	1	..	..	..	..	1	1	..	..	..	1	..	..	..	1	..	1	1	..	1	..	..	x	x	x	x	x	x	x	x	x
4	..	..	..	..	..	..	..	...	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
5	x	..	..	..	..	x	x	P	3	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	5	1	..	..	x	..	x	..	..	..	..	x	x	x	x
6	..	..	x	..	..	x	x	C	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	1	..	..	..	x	..	..	..	..	..	..	..	..
7	..	x	x	..	x	..	..	V	4	..	1	3	..	..	2	..	..	..	..	..	..	..	..	..	3	..	..	..	1	..	x	x	x	x	x	x	x	x	x
8	x	..	..	..	x	..	..	C	3	..	..	1	1	1	3	..	1	..	..	..	..	..	2	..	2	1	1	1	1	..	x	x	x	..	x	x	x	..	..
9	x	..	x	..	..	x	x	C	9	..	1	6	1	1	1	..	..	..	..	..	..	..	1	..	2	..	..	..	..	..	x	..	..	..	x	x	x	x	x
10	x	..	x	..	..	x	x	P	3	..	..	2	..	1	3	..	..	..	..	2	1	..	..	..	3	1	2	1	1	..	x	x	x	x	x	x	x	x	x
11	x	..	..	..	..	..	..	P	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	x	..
12	x	..	..	..	..	x	x	P	2	2	..	1	..	..	1	..	..	..	..	..	..	..	1	..	2	1	..	..	x	..	x	x	x	x	..	x			
13	x	x	..	..	..	x	x	V	2	..	..	1	..	1	2	..	..	..	1	..	1	..	..	..	4	1	1	1	1	..	x	x	x	x	x	x	x	x	x
14	x	..	..	..	x	..	..	P	6	2	..	3	..	1	6	..	1	..	..	4	1	..	..	..	2	2	..	2	2	..	x	x	..	x	x	x	x	..	x
15	..	..	..	..	..	..	..	P	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	..	..	x	..	x	..	x	x	..	..	..
16	..	..	..	..	..	x	x	P	4	..	..	1	..	3	3	1	1	..	..	..	1	..	1	..	4	1	1	1	..	..	x	x	x	x	x	x	x	x	x
17	..	..	..	..	..	x	x	VP	2	..	..	1	..	1	2	..	..	..	1	..	1	..	1	..	1	..	..	1	..	..	x	x	..	..	x	x	x	x	x
18	..	..	..	..	..	x	x	P	..	..	..	..	..	1	1	..	..	..	..	..	..	..	1	..	2	..	..	..	..	..	x	..	..	x	x	x	x	..	x
19	x	..	..	..	..	x	x	VP	5	2	..	2	..	1	..	..	..	1	..	..	1	..	..	..	3	2	..	2	1	..	x	x	x	x	x	x	x	x	x
20	..	..	..	..	..	x	x	P	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	1	..	..	..	x	..	x	x	x	x	x	..	x
21	x	..	..	..	x	..	..	P	2	..	1	..	1	..	2	..	..	..	..	..	..	..	..	..	3	1	..	1	1	1	x	x	x	..	..	x	x	..	x
22	x	..	..	..	..	x	x	P	2	..	..	2	..	..	1	..	..	..	1	..	..	..	..	..	3	1	3	1	1	..	x	x	x	x	x	x	x	x	x
23	..	x	..	..	x	..	..	V	19	4	4	6	1	4	10	..	..	2	..	..	1	..	..	5	10	5	3	4	1	5	x	x	x	x	x	x	x	x	x
24	x	..	..	..	..	..	..	C	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	1	..	1	..	x	x	x	..	..	..	..	..	..
25	x	..	..	x	..	x	x	P	4	1	..	2	..	1	3	..	1	1	..	..	..	..	..	1	2	2	2	..	..	1	x	x	x	x	x	x	x	x	x
26	..	..	..	..	..	x	x	P	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	1	1	..	..	..	x	x	x	x	x	x	x	..	x
27	x	..	..	..	..	x	x	P	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	2	1	..	..	..	1	x	x	x	..	x	x	x	x	x
28	x	..	x	x	..	x	x	P	3	..	..	..	..	3	3	..	1	..	..	..	2	..	..	..	2	1	1	1	1	..	x	x	x	x	x	x	x	x	x
29	..	x	..	..	x	..	..	V	1	..	..	1	..	..	1	..	..	..	..	..	..	..	..	..	2	2	..	1	..	..	x	x	x	x	x	x	x	..	x
30	..	x	..	..	..	x	x	V	2	..	..	2	..	..	2	..	..	..	1	..	..	..	..	1	2	1	1	1	1	1	x	x	x	x	x	x	x	x	x
31	x	..	..	..	..	..	..	C	..	1	..	1	..	1	1	1	..	..	..	..	1	..	..	..	1	..	..	..	..	..	x	..	x	x	x	x	x	x	x
32	x	..	..	..	..	x	x	VP	10	5	..	4	..	1	6	..	..	2	1	..	..	..	2	..	2	1	..	1	..	..	x	x	x	..	x	x	x	x	x
33	x	..	..	..	x	..	x	VP	4	..	..	2	..	2	3	1	1	..	1	..	..	..	1	..	6	1	3	1	..	1	x	x	x	x	x	x	x	x	x
34	..	x	..	..	x	..	..	V	4	..	..	3	..	1	4	..	..	..	..	1	1	..	1	..	6	4	2	2	1	..	x	x	x	x	x	x	x	x	x
35	x	..	.	..	..	x	x	P	2	..	2	..	..	..	2	..	..	..	..	..	..	..	1	..	1	1	1	..	..	..	x	x	x	x	x	..	..	..	x
36	..	x	..	x	x	..	..	P	1	..	..	1	..	..	1	..	..	..	..	1	..	..	..	1	1	1	1	1	..	..	x	x	x	x	x	x	x	x	x
37	x	..	..	..	..	x	x	P	5	..	1	..	..	2	1	2	..	..	..	1	..	..	2	..	2	1	2	3	1	1	x	x	x	..	x	x	x	..	x
38	..	x	..	..	x	..	..	P	1	..	..	1	..	..	1	..	..	..	1	..	..	..	1	..	2	2	1	1	..	..	x	x	x	x	x	x	x	x	x
39	x	x	..	..	..	x	x	VP	9	..	1	5	..	..	2	1	..	..	2	..	..	..	..	..	5	3	1	3	1	4	x	x	x	x	..	..	x	x	x
40	x	..	..	..	..	x	x	P	4	..	2	2	..	..	3	..	1	..	..	..	..	..	1	..	2	1	..	1	1	1	x	x	x	x	x	x	x	x	x

| | | |

Number of Center	Medical Supervision: Oto. C	Medical Supervision: Oto. R	Medical Supervision: Phys. C	Medical Supervision: Phys. R	Referrals: Med. Ex.	Referrals: Med. P	Referrals: Med. O	Restrictions	Personnel: Total Number	Degrees: B.A.	Degrees: B.S.	Degrees: M.A.	Degrees: M.S.	Degrees: Ph.D.	Certification: Audiology: M	Certification: Audiology: F	Certification: Audiology: AH	Certification: Audiology: AHP	Certification: Audiology: BH	Certification: Audiology: BHP	Certification: Speech: AS	Certification: Speech: ASP	Certification: Speech: BS	Certification: Speech: BSP	Equipment: Pure-Tone Aud.	Equipment: Speech Aud.	Equipment: Sound Rooms: S	Equipment: Sound Rooms: D	Equipment: PGSR	Equipment: Special	Services: Pure-Tone Audio.	Services: Speech Audio.	Services: H/A Selection	Services: H/A Demon.	Services: Lipreading	Services: Auditory Train.	Services: Speech Train.	Services: Psych. Testing	Services: Counsel. and Guid.
91	..	x	..	..	x	..	..	H	10	5	..	3	..	..	7	..	..	1	..	..	..	..	1	..	9	2	4	2	1	..	x	x	x	x	x	x	x	x	..
92	x	x	..	..	..	x	x	P	12	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	1	1	..	..	..	x	x	x	x	..	..	x	x	..
93	..	..	..	..	..	..	..	P	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	x	..	..	..	x	x	x	x	x	x	..	..	..
94	x	x	..	x	x	x	x	P	4	2	..	2	..	..	3	..	..	..	..	..	1	..	..	2	2	1	..	2	..	..	x	x	x	x	x	x	x	x	..
95	..	x	..	..	x	..	..	P	3	..	1	2	..	..	1	..	..	..	..	..	..	..	..	..	3	2	3	1	1	..	x	x	x	..	x	x	x	x	x
96	..	x	..	..	..	..	..	P	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6	3	2	..	1	..	x	x	x	..	..	..	..	..	..
97	..	x	..	..	x	..	..	H	1	..	..	1	..	..	1	..	..	1	..	..	..	..	..	..	4	1	2	1	1	..	x	x	x	x	x	x	x	x	x
98	x	..	..	..	..	x	x	VP	10	4	..	6	..	..	2	..	..	..	..	..	..	..	1	..	4	1	2	1	1	..	x	x	..	x	x	x	x	x	x
99	x	..	..	..	..	x	x	P	11	3	..	5	1	2	7	1	..	..	..	..	2	..	2	..	4	..	1	..	..	..	x	x	x	x	x	x	x	x	x
100	x	..	..	..	..	x	x	P	3	..	..	1	..	..	1	..	..	1	..	..	1	..	..	..	2	1	..	1	..	..	x	x	x	..	x	x	x	..	x
101	..	x	x	..	..	x	x	VP	8	3	..	2	1	2	4	1	..	..	..	..	2	..	3	..	8	3	4	2	1	3	x	x	x	x	x	x	x	x	x
102	x	..	..	..	..	x	x	P	5	..	1	2	1	..	2	..	..	..	..	..	..	..	..	..	3	2	..	2	1	2	x	x	x	x	x	x	x	..	x
103	x	..	..	..	..	..	..	P	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	1	x	..	x	..	x	x	x	x	..	..	..	..	x
104	..	..	x	..	..	x	x	VP	4	..	..	..	1	3	3	1	..	..	..	..	1	..	..	..	3	..	1	..	..	..	x	x	x	x	x	x	x	x	x
105	..	..	..	..	..	x	x	VP	5	2	..	2	..	1	2	..	..	..	..	..	..	1	1	..	4	..	2	..	..	1	x	x	x	x	x	x	x	x	x
106	x	..	x	..	..	x	x	VP	4	..	..	1	1	..	2	..	..	..	1	..	..	..	..	1	2	2	..	1	1	..	x	x	x	x	x	x	x	x	x
107	x	..	x	..	..	x	x	VP	23	5	..	14	..	4	8	1	..	1	..	1	2	1	4	..	7	4	2	3	..	10	x	x	x	x	x	x	x	x	x
108	x	..	x	..	..	x	x	P	4	..	..	..	1	..	1	..	..	..	..	..	..	..	..	..	2	1	..	1	..	..	x	x	..	x	x	x	x	..	x
109	..	..	x	..	..	x	x	C	4	1	..	3	..	..	1	..	..	..	4	..	..	..	..	..	18	2	..	1	..	..	x	x	..	x	x	x	x	..	x
110	x	..	..	..	..	x	x	VP	2	..	..	..	..	2	2	..	1	..	1	..	1	..	..	..	8	..	4	1	..	1	x	x	..	..	..	..	..	x	..
111	..	..	x	x	..	x	x	P	4	..	..	4	..	..	4	..	..	1	..	1	..	2	..	..	3	1	2	..	1	..	x	x	x	x	x	x	x	x	x
112	..	..	x	..	..	x	x	P	4	3	..	1	..	..	3	..	..	..	1	..	..	2	..	..	3	1	1	..	1	..	x	x	x	x	x	x	x	x	x
113	x	..	..	..	..	x	x	VP	3	..	..	3	..	..	..	..	..	..	1	..	..	..	2	..	1	1	..	1	..	..	x	x	x	x	x	x	x	x	x
114	x	..	..	..	..	x	x	VP	14	..	..	5	..	4	6	..	2	..	1	..	2	..	1	..	7	..	2	2	..	..	x	x	x	x	x	x	x	x	x
115	x	..	..	..	..	x	x	VP	3	..	..	2	..	..	1	..	..	..	1	..	..	..	..	..	1	1	1	1	1	..	x	x	x	x	x	x	x	x	x
116	x	..	..	..	..	..	..	P	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	1	..	..	..	x	x	..	x	x	x	x	x	x
117	x	..	..	..	..	x	x	P	8	..	1	4	1	1	..	..	..	..	2	..	..	..	..	..	1	1	1	..	..	..	x	x	..	x	x	x	x	x	x
118	x	..	x	..	..	x	x	P	2	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4	1	..	1	1	1	x	x	x	x	x	x	x	x	x
119	..	..	..	..	..	x	x	P	1	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..	..	..	x	x	x	x	x	x	x	x	x
120	x	..	..	..	..	x	x	VP	2	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	x	x	x	x	x	x	x	x	x	x	x	x	x
121	x	..	..	..	..	..	..	P	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	..	1	1	..	..	..	..	x	x	x	x	x	x	x	x	x
122	..	..	..	..	..	x	x	C	2	..	..	..	1	..	1	..	..	1	..	..	..	..	..	..	2	1	..	1	1	..	x	x	x	x	..	x	x	x	x
123	x	..	..	..	..	..	..	VP	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	1	..	..	..	x	x	x	x	..	..	..	x	x
124	x	..	..	..	..	..	..	V	..	3	..	2	..	..	..	..	..	1	..	..	..	..	..	..	1	1	1	..	..	..	x	x	x	x	x	x	x	x	x
125	x	..	..	..	..	x	x	P	4	..	..	4	..	..	1	..	..	..	1	..	..	..	..	..	4	2	2	..	1	1	x	x	x	x	x	x	x	x	x
126	..	x	..	..	..	x	x	VP	9	2	..	5	..	2	8	1	4	..	2	..	..	..	1	..	1	1	1	1	..	..	x	x	x	x	x	x	x	..	x
127	x	..	..	..	..	..	..	P	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	9	2	2	2	4	2	x	x	x	x	x	x	..	x	x
128	..	..	..	..	..	x	x	C	4	..	..	2	2	..	4	..	..	..	..	..	..	..	..	..	1	1	1	..	..	..	x	x	x	x	x	x	x	x	x
129	..	..	x	..	..	x	x	P	14	1	4	..	5	4	8	1	..	..	2	2	..	..	..	..	5	..	..	..	..	..	x	..	..	x	x	x	x	x	x
130	x	..	..	..	..	x	x	P	1	..	..	1	..	..	1	..	..	..	..	..	3	..	3	..	4	1	1	1	..	..	x	x	x	x	x	x	x	x	x
131	x	..	..	..	x	..	..	P	3	..	..	2	..	1	3	..	..	..	..	..	..	..	..	1	1	1	1	1	..	..	x	x	x	x	x	x	x	..	x
132	x	..	x	..	x	..	..	VP	6	2	..	3	..	..	4	..	1	..	..	..	1	1	..	..	4	..	1	..	..	..	x	x	x	..	x	x	x	x	x
133	..	..	x	..	x	..	..	VP	13	..	..	7	3	3	10	..	1	..	..	..	1	..	4	..	8	2	1	1	1	2	x	x	x	..	x	x	x	..	x
134	..	..	x	..	..	x	x	VP	7	..	..	5	..	2	6	..	..	..	..	1	1	..	2	1	2	1	1	1	1	..	x	x	x	x	x	x	x	x	x
135	..	..	x	..	..	x	x	P	1	..	..	..	..	1	1	..	..	..	..	..	1	..	..	..	1	1	..	..	..	..	x	..	..	x	x	x	x	x	x
136	x	..	..	..	x	..	..	VP	4	..	1	1	..	..	3	..	..	..	1	..	..	..	1	..	3	2	..	1	1	1	x	x	x	x	x	x	x	x	x
137	x	..	..	..	..	x	x	VP	8	..	1	..	5	2	8	..	1	..	..	..	1	..	3	..	4	1	..	2	1	2	x	x	x	x	x	x	x	..	x
138	x	..	..	..	..	x	x	VP	11	..	5	..	3	1	5	..	1	..	..	..	..	..	..	..	1	1	1	1	1	..	x	x	x	x	x	x	x	x	x
139	x	..	x	..	..	x	x	C	2	1	..	1	..	..	2	..	..	..	..	..	1	..	..	..	1	1	x	x	x	x	x	x	x	x	x	x	x	x	x
140	..	x	..	..	..	x	x	P	5	2	..	..	..	3	3	..	..	1	..	..	2	..	1	..	1	1	..	..	1	..	x	x	x	..	x	x	x	x	x
141	..	x	..	..	..	x	x	VP	3	1	1	1	..	..	1	..	..	..	..	..	..	..	..	..	5	1	4	1	1	..	x	x	x	x	x	x	x	x	x
142	..	..	x	..	..	x	x	VP	5	2	..	2	..	..	4	..	..	..	..	..	..	..	1	..	4	1	2	1	1	..	x	x	x	x	x	x	x	x	x
143	..	..	..	..	..	..	x	P	5	1	..	1	..	3	4	1	1	..	..	2	..	..	1	1	1	1	1	1	..	..	x	x	x	x	x	x	x	..	x
144	..	..	..	..	..	x	x	VP	2	..	..	1	..	1	2	1	..	..	..	..	2	..	1	..	5	2	..	2	1	..	x	x	x	x	x	x	x	x	x
145	..	..	..	..	..	..	..	C	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	1	3	1	1	1	1	..	x	x	x	..	x	x	x	x	x
146	x	..	..	..	..	x	x	VP	2	..	1	2	..	..	1	..	1	..	..	..	..	..	1	..	4	1	10	..	..	..	x	..	..	..	..	..	x	..	x

Listing in this survey does not constitute endorsement by any organization for the services rendered by the center. Any changes or corrections in listing should be sent to The Audiology Foundation, Box 21, Glenview, Ill.

Detailed information in regard to hearing rehabilitation centers listed below may be found in the Table.

ESTABLISHED HEARING REHABILITATION CENTERS

ALABAMA

1. Talladega, Alabama Hearing and Speech Center, Box 268
   Auspices: Alabama Institute for Deaf and Blind
   Administrative Head: John E. Bryan, L.H.D., President
   Medical Supervisors: G. E. Fisher, M.D.; J. L. Hicks, M.D.
2. Tuscaloosa, Speech and Hearing Clinic, Box 2025, University of Alabama
   Auspices: University of Alabama
   Administrative Head: T. Earle Johnson, Ph.D., Head, Department of Speech, AH
   Medical Supervisor: University Hospital and Medical College of Alabama Staff

ARIZONA

3. Tempe, Norman E. Iverson Speech and Hearing Center, P. O. Box 506
   Auspices: Private
   Administrative Head: Norman E. Iverson, Ph.D., BS, BH
   Medical Supervisor: Application pending to Arizona Medical Association

ARKANSAS

4. Little Rock, Audiology Clinic, Veterans Administration Regional Office, 555 Building, 211 Broadway
   Auspices: Veterans Administration

CALIFORNIA

5. Alhambra, Children's Hearing and Speech Clinic, 612 W. Shorb
   Auspices: National Auricular Foundation, Inc.
   Administrative Head: Willard B. Hargrave, Executive Vice-President
   Medical Supervisor: M. E. Trainor, M.D.
6. Bakersfield, Crippled Children Services Clinics, P. O. Box 997
   Auspices: Health Department
7. Los Angeles, Audiology and Speech Correction Clinic, Room 804, 1031 S. Broadway
   Auspices: Veterans Administration
   Administrative Head: Edward W. Gibbons, M.A., Chief
   Medical Supervisor: Yes
8. Los Angeles, Hearing and Speech Clinic, 4614 Sunset Blvd.
   Auspices: Children's Hospital
   Administrative Head: Jacqueline Keaster, M.S., AH
   Medical Supervisor: Victor Goodhill, M.D.
9. Los Angeles, John Tracy Clinic, 806 W. Adams Blvd.
   Auspices: Nonprofit corporation
   Administrative Head: Mrs. Spencer Tracy, Director (Edgar L. Lowell, Ph.D., Administrator)
   Medical Supervisor: Victor Goodhill, M.D., Consultant
10. Los Angeles, Speech-Hearing Clinic, University of Southern California, 930 W. 37th St.
    Auspices: University of Southern California
    Administrative Head: Victor P. Garwood, Ph.D., AS, BHP
    Medical Supervisor: Victor Goodhill, M.D., Consultant
11. Pasadena, Pasadena Dispensary, 38 Congress St.
    Auspices: Nonsectarian board of directors
    Administrative Head: Florence E. King, Superintendent, F.A.C.H.A.
    Medical Supervisor: David T. Proctor, M.D.

12. San Diego, San Diego Hearing Society, 3843 Herbert St.
Auspices: Community Chest
Administrative Heads: Mrs. Vesta Muehleisen, A.B., Executive Secretary; Miss Louise P. Feldman, B.A., M.A., Audiologist
Medical Supervisor: Phillip S. Mountjoy, M.D., Consultant

13. San Francisco, Audiology and Speech Correction Clinic, Veterans Administration Hospital, 42nd and Clement Sts. (Fort Miley)
Auspices: Veterans Administration
Administrative Head: Richard A. Winchester, Ph.D., ASP
Medical Supervisor: Walter P. Work, M.D., Consultant

14. San Francisco, San Francisco Hearing and Speech Center, 1609 Scott St.
Auspices: San Francisco Medical Society and Mount Zion Hospital
Administrative Head: Hayes A. Newby, Ph.D., AH
Medical Supervisor: Committee of four otologists; Robert C. McNaught, M.D., Chairman

15. San Francisco, San Francisco Hearing Society, 2015 Steiner St.
Auspices: Community Chest
Administrative Head: Carolyn A. Donworth, Executive Director
Medical Supervisor: None

16. Stanford, Stanford Speech and Hearing Clinic, Stanford University
Auspices: Stanford University
Administrative Head: Virgil A. Anderson, Ph.D., AS
Medical Supervisor: Community Advisory Board

17. Stockton, College of the Pacific Speech and Hearing Center, Pacific Ave.
Auspices: College of the Pacific
Administrative Head: Howard L. Runion, Ph.D., AS
Medical Supervisor: None

18. Whittier, Speech and Hearing Clinic, Whittier College
Auspices: Whittier College
Administrative Head: Lester L. Harris, Ph.D., Director, BS
Medical Supervisor: None

COLORADO

19. Denver, University of Denver Hearing Center, 1445 Cleveland Place
Auspices: University of Denver
Administrative Head: W. J. Clark, Ph.D., Director, Speech and Hearing Center, AHP
Medical Supervisor: Colorado Otolaryngology Society Committee; Robert C. Shattuck, M.D., Chairman

CONNECTICUT

20. Hartford, Hartford Hearing League, Inc., 252 Asylum St.
Auspices: Private social agency with Community Chest support
Administrative Head: Mrs. Helen S. Lowry, A.B., Executive Secretary
Medical Supervisor: None

21. New Haven, Hearing and Speech Center of Yale-New Haven Medical Center
Auspices: Grace-New Haven Community Hospital and Yale School of Medicine
Administrative Head: Harris I. Pomerantz, M.S.
Medical Supervisor: John A. Kirchner, M.D.

DELAWARE

22. Wilmington, Audiology and Speech Center, Delaware Hospital, 501 W. 14th St.
Auspices: Private General Hospital, Department of Otolaryngology
Administrative Head: L. LeRoy Horne, M.A., BH
Medical Supervisor: Emil Mayerberg, M.D.

DISTRICT OF COLUMBIA

23. Washington, D. C., Audiology and Speech Correction Center, Forest Glen Section, Walter Reed Army Hospital
Auspices: Department of the Army
Administrative Head: James P. Albrite, Major (MC), M.D.
Medical Supervisor: James P. Albrite, Major (MC), M.D.

24. Washington, D. C., The Children's Hospital Hearing Clinic, 13th and W Sts., N.W.
    Auspices: The Children's Hospital
    Administrative Head: Mrs. Edna K. Monsees, Audiologist
    Medical Supervisor: Allan E. Walker Jr., M.D.
25. Washington, D. C., Hearing and Speech Clinic, 1147 15th St., N.W.
    Auspices: Episcopal Eye, Ear and Throat Hospital
    Administrative Head: Deaconess Margaret Bechtol
    Medical Supervisor: Frasier Williams, M.D.
26. Washington, D. C., Hearing Rehabilitation Center, 1911 R St., N.W.
    Auspices: Private
    Administrative Head: Mrs. Edna K. Monsees, Director
    Medical Supervisor: Referring otologists
27. Washington, D. C., Washington Hearing Society, 1934 Calvert St., N.W.
    Auspices: Community Chest, Board of Directors, and Committee of Otolaryngologists
    Administrative Head: Ruth A. Hudnut, M.A.
    Medical Supervisor: Committee of Otolaryngologists

FLORIDA

28. Gainesville, Speech and Hearing Clinic, University of Florida
    Auspices: University of Florida
    Administrative Head: McKenzie Buck, Ph.D., AS
    Medical Supervisor: Dekle Taylor, M.D., Jacksonville, Fla.
29. Pass-A-Grille (St. Petersburg), Audiology and Speech Correction Clinic, Veterans Administration Regional Office
    Auspices: Veterans Administration
    Administrative Head: Robert D. Bitler, M.A., M
    Medical Supervisor: August E. Gehrke, M.D.

GEORGIA

30. Atlanta, Audiology and Speech Correction Clinic, Veterans Administration Regional Office, 105 Pryor St., N.E.
    Auspices: Veterans Administration
    Administrative Head: Allan J. Heffler, M.A., Chief, BH
    Medical Supervisor: C. A. Almand, M.D., Chief Medical Officer
31. Atlanta, Davison School of Speech Correction, 1780 N. Decatur Rd., N.E.
    Auspices: Private
    Administrative Head: Mrs. W. W. Davison, A.B., Director, AS
    Medical Supervisors: Lester Brown, M.D.; Calhoun McDougal, M.D., etc.
32. Atlanta, Junior League School for Speech Correction, 2020 Peachtree Rd., N.W.
    Auspices: Junior League of Atlanta, Inc.
    Administrative Head: Claude S. Hayes, Ph.D., Director of Education, AHP
    Medical Supervisors: B. Russell Burke, M.D.; Lester Brown, M.D.; William Warren, M.D.

ILLINOIS

33. Champaign, University Hearing Center, 321 Illini Hall, 6th and John Sts.
    Auspices: University of Illinois
    Administrative Head: E. Thayer Curry, Ph.D., Director, AH
    Medical Supervisors: Otologists in the area serve as consultants
34. Chicago, Audiology and Speech Correction Clinic, West Side Hospital, 820 S. Damen Ave.
    Auspices: Veterans Administration
    Administrative Head: Martin Cohen, M.A., BS
    Medical Supervisor: Nathaniel Van Dorf, M.D.
35. Chicago, Chicago Hearing Society, 30 W. Washington St.
    Auspices: Voluntary Social Agency; partial support from Chicago Community Fund
    Administrative Head: Miss Mary L. Thompson, M.A., Executive Secretary
    Medical Supervisor: Consultant
36. Chicago, Hearing and Speech Service, 29th St. and Ellis Ave.
    Auspices: Michael Reese Hospital

Administrative Head: Peter A. Campanelli, M.A.
Medical Supervisor: Robert Henner, M.D.

37. Chicago, Hearing Clinic, Northwestern University Medical School, 303 E. Chicago Ave.
Auspices: Northwestern University, Ear, Nose and Throat Department
Administrative Head: George E. Shambaugh Jr., M.D.
Medical Supervisor: George E. Shambaugh Jr., M.D.

38. Chicago, Presbyterian Hospital Speech and Hearing Center, 1753 W. Congress St.
Auspices: Presbyterian Hospital
Administrative Head: Duncan R. C. Scott, M.A., BS, BH
Medical Supervisor: Stanton A. Friedberg, M.D.

39. Chicago, Speech and Hearing Rehabilitation Center, Illinois Eye and Ear Infirmary, 904 W. Adams
Auspices: University of Illinois, College of Medicine
Administrative Head: Francis L. Lederer, M.D.
Medical Supervisor: Francis L. Lederer, M.D.

40. Chicago, St. Luke's Speech and Hearing Service, 1439 S. Michigan Ave.
Auspices: St. Luke's Hospital, Department of Otolaryngology
Administrative Head: William F. Waldrop, M.A., AH
Medical Supervisor: Arthur Coombs, M.D.

41. Chicago, University of Chicago Clinics, 950 E. 59th St.
Auspices: University of Chicago School of Medicine, Department of Otolaryngology
Administrative Heads: R. F. Naunton, M.B.B.S.; Joseph Wepman, Ph.D., AS
Medical Supervisor: J. R. Lindsay, M.D.

42. Evanston, Northwestern University Hearing Clinics, Adult
Auspices: Northwestern University
Administrative Head: John H. Gaeth, Ph.D., AH
Medical Supervisors: George E. Shambaugh Jr., M.D.; Eugene Derlacki, M.D.

43. Evanston, Children's Hearing and Aphasia Clinic, Speech Annex, Northwestern University
Auspices: School of Speech, Northwestern University
Administrative Head: Helmer R. Myklebust, Ed.D., Director
Medical Supervisor: George E. Shambaugh Jr., M.D.

44. Normal, Hearing Laboratory, Special Education Building, Illinois State Normal University
Auspices: Illinois State Normal University
Administrative Head: Glenn Taylor, Ph.D., AH
Medical Supervisor: C. W. Hardy, M.D.

45. Peoria, Bradley University Speech Clinic
Auspices: Bradley University
Administrative Head: Clara Mawhinney, Ph.D., AH
Medical Supervisors: Morris H. Cohen, M.D.; Robert Hart, M.D.

46. Rockford, Rockford College Speech and Hearing Center, Seminary St.
Auspices: Rockford College
Administrative Head: Mildred F. Berry, Ph.D., F
Medical Supervisor: None

INDIANA

47. Bloomington, Indiana University Speech and Hearing Center, University Clinics Building
Auspices: Indiana University
Administrative Head: Robert Milisen, Ph.D., F, AS
Medical Supervisor: Bryan Quarles, M.D.

48. Indianapolis, Audiology and Speech Clinic, Indiana University Medical Center, 1100 W. Michigan St.
Auspices: Department of Otorhinolaryngology
Administrative Head: Francis L. Sonday, A.B., Director, AH
Medical Supervisors: Marlow W. Manion, M.D.; David E. Brown, M.D.

49. Lafayette, Purdue University Speech and Hearing Clinic
Auspices: Purdue University
Administrative Head: M. D. Steer, Ph.D., AS
Medical Supervisors: Consultants: R. Calvert, M.D.; E. L. Van Buskirk, M.D.; etc.

50. Muncie, Out-Patient (Speech and Hearing), Ball State Teachers College
    Auspices: Ball State Teachers College
    Administrative Head: Alan W. Huckleberry, Ed.D.
    Medical Supervisor: Amelia T. Wood, M.D.
51. South Bend, Hearing Society of St. Joseph County, 206 Income Building. 511 W. Colfax
    Auspices: Community Chest
    Administrative Head: Leva M. Ritter, B.S
    Medical Supervisor: None
52. Terre Haute, Special Education Clinic
    Auspices: Indiana State Teachers College
    Administrative Head: Rutherford B. Porter, Ed.D.
    Medical Supervisor: Floyd Riggs, M.D.

IOWA

53. Iowa City, University Hospitals, Department of Otolaryngology
    Auspices: State University of Iowa, College of Medicine
    Administrative Head: D. M. Lierle, M.D., M.S., D.Sc., F
    Medical Supervisor: D. M. Lierle, M.D.

KANSAS

54. Kansas City, University of Kansas Medical Center, 39th St. and Rainbow Blvd.
    Auspices: University of Kansas Medical Center Board of Regents
    Administrative Head: June Miller, M.A., AH
    Medical Supervisor: L. B. Spake, M.D.
55. Wichita, Institute of Logopedics, 2400 Jardine Dr.
    Auspices: Nonprofit corporation
    Administrative Head: Martin F. Palmer, Sc.D., Director, AS
    Medical Supervisor: C. T. Hinshaw, M.D.

MAINE

56. Portland, Portland Hearing Society, 653A Congress St.
    Auspices: American Hearing Society
    Administrative Head: Pauline G. Carter, B.A., Executive Secretary
    Medical Supervisor: None
57. Waterville, Speech and Speech-Reading Clinic, Thayer Hospital, North St.
    Auspices: Department of Health and Welfare and Thayer Hospital
    Administrative Head: Elizabeth O. Koons, M.A., Instructor
    Medical Supervisor: Frederick I. Hill, M.D.

MARYLAND

58. Baltimore, Baltimore Hearing Society, Inc., 322 N. Charles St.
    Auspices: Community Chest
    Administrative Head: Eugene L. Morrill Jr., M.Ed., Executive Director, M
    Medical Supervisor: None
59. Baltimore, Johns Hopkins Hearing and Speech Center, Johns Hopkins Hospital
    Auspices: Johns Hopkins School of Medicine
    Administrative Head: William G. Hardy, Ph.D., AH
    Medical Supervisor: John E. Bordley, M.D.

MASSACHUSETTS

60. Boston, Boston Guild for the Hard of Hearing, 283 Commonwealth Ave.
    Auspices: United Community Services
    Administrative Head: Mrs. Claire K. Kennedy, Executive Director
    Medical Supervisor: Committee of consulting otologists supplied by
    Otolaryngological Association
61. Boston, Boston University Speech and Hearing Center, 332 Bay State Rd.
    Auspices: Boston University
    Administrative Head: Wilbert Pronovost, Ph.D., AS
    Medical Supervisors: Four consultant otologists

62. Boston, Boston VA Audiology and Speech Correction Clinics, 17 Court St.
Auspices: Veterans Administration
Administrative Head: George E. Hill, M.A., Chief Audiologist
Medical Supervisor: Benjamin Bell, M.D.

63. Boston, Samuel D. Robbins Speech and Hearing Clinic of Emerson College, 145 Beacon St.
Auspices: Emerson College Women's Committee
Administrative Head: Catherine C. Perry, A.M., AS
Medical Supervisors: Members of Advisory Board

64. Boston, Hearing Clinic, 300 Longwood Ave.
Auspices: Children's Medical Center
Administrative Head: Adam J. Sortini, M.A., BS, BH
Medical Supervisor: Carlyle G. Flake, M.D.

65. Boston, Institute for Speech Correction, 419 Boylston St.
Auspices: Board of Trustees
Administrative Head: Samuel D. Robbins, A.M., AS
Medical Supervisor: Gale C. Perry, M.D.

66. Boston, Winthrop Foundation for Investigation of Deafness, Massachusetts Eye and Ear Infirmary, 243 Charles St.
Auspices: Massachusetts Eye and Ear Infirmary
Administrative Head: Alan S. Feldman, M.S.
Medical Supervisor: Philip E. Meltzer, M.D.

67. Springfield, Springfield Hearing League, Inc., 1694 Main St.
Auspices: United Fund, Inc., of Springfield
Administrative Head: Mrs. Grace F. Gaffney, A.B.
Medical Supervisor: None

68. Worcester, Worcester Hearing League, 306 Main St.
Auspices: Community Chest and Chapter of American Hearing Society
Administrative Head: Miss Kathleen Kirby, B.A., Executive Director
Medical Supervisor: None

MICHIGAN

69. Ann Arbor, University of Michigan Speech Clinic, 1007 E. Huron
Auspices: University of Michigan Speech Department and Institute for Human Adjustment
Administrative Head: H. H. Bloomer, Ph.D., F, AS
Medical Supervisor: L. Dell Henry, M.D.

70. Detroit, Detroit League for the Handicapped, 1401 Ash St.
Auspices: United Community Services
Administrative Head: Robert W. Will, M.Ed.
Medical Supervisor: None

71. Detroit, Henry Ford Hospital Audiology and Speech Clinic
Auspices: Henry Ford Hospital
Administrative Head: A. Bruce Graham, Ph.D., AHP
Medical Supervisor: J. Lewis Dill, M.D.

72. Detroit, Wayne University Speech and Hearing Clinic, 656 W. Warren
Auspices: Wayne University
Administrative Head: George Kopp, Ph.D., AS
Medical Supervisor: None

73. East Lansing, Hearing Clinic, Michigan State College
Auspices: Speech Department, Michigan State College
Administrative Head: Charles Pedrey, Ph.D.
Medical Supervisor: Pending

74. Flint, Michigan School for the Deaf, Court St.
Auspices: State Board of Education
Administrative Head: Thomas H. Poulos, M.A., BH
Medical Supervisor: None

75. Kalamazoo, Constance Brown Society for Better Hearing, 316 Commerce Building
Auspices: Kalamazoo Foundation
Administrative Head: Alfred R. Thea, M.S.
Medical Supervisor: Ralph B. Fast, M.D.

76. Lansing, Michigan Association for Better Hearing, 408 Hollister Building
Auspices: United Health and Welfare Fund of Michigan, Inc.
Administrative Head: Stahl Butler, M.A., Executive Director
Medical Supervisors: Three otolaryngologists on Advisory Board

MINNESOTA

77. Minneapolis, Audiology Clinic, University of Minnesota Hospitals
Auspices: University of Minnesota
Administrative Head: Frank M. Lassman, Ph.D., AH, AS
Medical Supervisor: Lawrence R. Boies, M.D.

78. Minneapolis, Speech and Hearing Clinic, University of Minnesota
Auspices: University of Minnesota
Administrative Head: Ernest H. Henrikson, Ph.D., AS
Medical Supervisor: George Tangen, M.D.

79. Rochester, Mayo Clinic Audiology Center, EENT Department
Auspices: Mayo Clinic
Administrative Head: Henry S. Williams, M.D.
Medical Supervisor: Henry S. Williams, M.D.

MISSISSIPPI

80. Jackson, Magnolia Speech and Hearing Foundation and Treatment Center, 519 Mississippi St.
Auspices: Mississippi Society for Crippled Children, United Cerebral Palsy, Jaycees, B.P.W.C., etc.
Administrative Head: George A. Falconer, M.S., AH
Medical Supervisor: None

MISSOURI

81. Columbia, University of Missouri Speech and Hearing Clinic, Switzler Hall
Auspices: University of Missouri
Administrative Head: Charlotte G. Wells, Ph.D., AS
Medical Supervisor: University of Missouri Student Health Service

82. Kansas City, Audiology Clinic, Veterans Administration Regional Office, 1828 Walnut
Auspices: Veterans Administration
Administrative Head: Mrs. Phyllis Weifenbach, M.S., M
Medical Supervisor: J. W. Findley, M.D.

83. St. Louis, Central Institute for the Deaf, 818 S. Kingshighway
Auspices: Private Board of Managers
Administrative Head: S. R. Silverman, Ph.D., AH
Medical Supervisors: Theo. E. Walsh, M.D.; Harold Cutler, M.D.

NEBRASKA

84. Lincoln, Speech and Hearing Laboratories, University of Nebraska
Auspices: University of Nebraska
Administrative Head: John Wiley, Ph.D., AS
Medical Supervisor: None

NEW JERSEY

85. Newark, Henry C. Barkhorn Memorial Hearing and Speech Center, 77 Central Ave.
Auspices: Newark Eye and Ear Infirmary
Administrative Head: Herbert E. Rickenberg, M.A., BS
Medical Supervisor: William H. Hahn, M.D.

86. Newark, St. Barnabas Hearing and Speech Center, 701 High St.
Auspices: Hospital of St. Barnabas and for Women and Children
Administrative Head: Lyndon A. Peer, M.D.
Medical Supervisors: Franklin Keim, M.D.; Louise Fischer, M.D.

87. Paterson, Paterson League for the Hard of Hearing, 182 Ellison St.
Auspices: Community Chest
Administrative Head: Samuel M. Cohen, Ed.M., BS
Medical Supervisor: None

NEW YORK

88. Albany, Albany Medical Center Conservation of Hearing Clinic, Albany Hospital
Auspices: Albany Hospital and New York State Health Department
Administrative Head: George J. Righter, M.D.
Medical Supervisor: George J. Righter, M.D.

89. Brooklyn, Apostolate for the Deaf and Hard of Hearing, 191 Joralemon St.
Auspices: Catholic Charities, Diocese of Brooklyn
Administrative Head: Rev. George J. Haye, B.A.
Medical Supervisor: None

90. New York, Audiology Clinic, Veterans Administration New York Regional Office, 252 Seventh Ave.
Auspices: Veterans Administration
Administrative Head: None at present
Medical Supervisor: Sylvester Daly, M.D.

91. New York, Hofheimer Speech and Hearing Clinic, 622 W. 168th St.
Auspices: Columbia-Presbyterian Medical Center
Administrative Head: Donald M. Markle, M.Ed., AHP
Medical Supervisor: Edmund P. Fowler Jr., M.D.

92. New York, Harlem Eye and Ear Hospital, 2099 Lexington Ave.
Auspices: Harlem Eye and Ear Hospital
Administrative Head: Eugen Grabscheid, M.D.
Medical Supervisor: John J. Levbarg, M.D.

93. New York, Hearing Rehabilitation, 330 E. 63rd St.
Auspices: Private
Administrative Head: Mary Wood Whitehurst, M.A.
Medical Supervisor: None

94. New York, New York University Bellevue Medical Center, Institute of Physical Medicine and Rehabilitation, 400 E. 34 St.
Auspices: Department of Otolaryngology, NYU Bellevue Medical Center
Administrative Head: Mrs. Martha L. Taylor, M.A., AS
Medical Supervisors: John Daly, M.D.; Roy Shults, M.D.

95. New York, Hearing and Speech Clinic, Manhattan Eye, Ear and Throat Hospital, 210 E. 64th St.
Auspices: Manhattan Eye, Ear and Throat Hospital
Administrative Head: Mary Wood Whitehurst, M.A., M
Medical Supervisor: Richard Bellucci, M.D.

96. New York, New York Eye and Ear Infirmary, Audiology and Hearing Research Department, 218 Second Ave.
Auspices: New York Eye and Ear Infirmary
Administrative Head: James A. DePew Jr.
Medical Supervisor: James Swift Hanley, M.D.

97. New York, Audiology Clinic, 525 E. 68th St.
Auspices: The New York Hospital - Cornell Medical Center
Administrative Head: Miss Margaret Soisson, M.A., AHP
Medical Supervisor: James A. Moore, M.D.

98. New York, New York League for the Hard of Hearing, 480 Lexington Ave.
Auspices:
Administrative Head: Estelle E. Samuelson
Medical Supervisor: Edmund Prince Fowler, M.D.

99. New York, Queens Speech and Hearing Center, Queens College (Flushing 67)
Auspices: Queens College
Administrative Head: Jon Eisenson, Ph.D., F, AS
Medical Supervisor: Otto Risch, M.D.

100. Rochester, Rochester Hearing and Speech Society, Inc., 800 Main St., East
Auspices: Community Chest
Administrative Head: Stanley Dickson, M.A., AHP
Medical Supervisor: C. Stewart Nash, M.D.

101. Syracuse, Gordon D. Hoople Hearing and Speech Center, 805 S. Crouse Ave.
Auspices: Syracuse University
Administrative Head: Louis M. DiCarlo, Ed.D., F
Medical Supervisor: Gordon D. Hoople, M.D.

102. Utica, Hearing and Speech Center, Children's Hospital Home, 1675 Bennett St.
Auspices: Children's Hospital Home, New York State Association for Crippled Children
Administrative Head: Karl Gruppe, M.D.
Medical Supervisor: Karl Gruppe, M.D.

NORTH CAROLINA

103. Winston-Salem, North Carolina Baptist Hospital and Bowman Gray School of Medicine, Audiology Clinic, S. Hawthorn St.
Auspices: Department of Otolaryngology
Administrative Head: J. A. Harrill, M.D.
Medical Supervisor: J. A. Harrill, M.D.

OHIO

104. Athens, Ohio University Speech and Hearing Clinic, Ohio University
Auspices: School of Dramatic Art and Speech, Ohio University
Administrative Head: A. C. LaFollette, Ph.D., AS
Medical Supervisor: Herndon Hudson, M.D.

105. Bowling Green, Speech and Hearing Clinic, Bowling Green State University
Auspices: Bowling Green State University
Administrative Head: Melvin Hyman, Ph.D., ASP
Medical Supervisor: None

106. Cincinnati, Cincinnati Speech and Hearing Center, 616 Walnut St.
Auspices: Community Chest
Administrative Head: Warren E. Johnson, M.A., Director, BH
Medical Supervisors: Samuel Seltz, M.D.; William Craddock, M.D.

107. Cleveland, The Cleveland Hearing and Speech Center, 11206 Euclid Ave.
Auspices: The Welfare Federation of Greater Cleveland and Western Reserve University
Administrative Head: George J. Fortune, M.A., Director
Medical Supervisor: Medical, Scientific and Research Committee of the Board of Trustees

108. Columbus, Columbus Hearing Society, 137 E. State St.
Auspices: Community Chest of Columbus and Franklin County
Administrative Head: Marshall A. Becker, M.A.
Medical Supervisor: Russel G. Means, M.D.

109. Columbus, Ohio Department of Health, Division of Child Hygiene, Ohio Departments Building
Auspices: Division of Child Hygiene, Ohio Department of Health
Administrative Head: Crayton Walker, M.A., AHP
Medical Supervisor: Margot D. Hartmann, M.D.

110. Columbus, Ohio State University Speech and Hearing Clinic
Auspices: Department of Speech, Ohio State University
Administrative Head: Henry M. Moser, Ph.D., F, AS
Medical Supervisor: None

111. Kent, Speech and Hearing Clinics, Kent State University
Auspices: Kent State University
Administrative Head: John R. Montgomery, M.A., AS
Medical Supervisor: A. O. DeWeese, M.D.

112. Toledo, Toledo Hearing League, 2313 Ashland Ave.
Auspices: Board of Trustees; private agency
Administrative Head: Mrs. George Reisbach
Medical Supervisor: None

113. Youngstown, Youngstown Hearing and Speech Center, 69 Illinois Ave.
Auspices: Youngstown Hearing Society
Administrative Head: May Vetterle, M.A., BH
Medical Supervisors: Committee of otologists

OKLAHOMA

114. Oklahoma City, Oklahoma University Speech and Hearing Clinic, Norman, Oklahoma and Crippled Children's Hospital, Oklahoma City
Auspices: University of Oklahoma
Administrative Head: John W. Keys, Ph.D., AH
Medical Supervisors: O. A. Watson, M.D.; Chester McHenry, M.D.

OREGON

115. Portland, Portland Center for Hearing and Speech, Inc., 220 S.W. Alder, Education Center Building, Room 402
Auspices: United Fund
Administrative Head: George J. Leshin, M.Ed.
Medical Supervisor: David D. DeWeese, M.D.

116. Portland, University of Oregon Medical School, Audiology Clinic, 3181 Sam Jackson Park Rd.
Auspices: University of Oregon Medical School
Administrative Head: David D. DeWeese, M.D.
Medical Supervisor: David D. DeWeese, M.D.

PENNSYLVANIA

117. Lancaster, The Hearing Conservation Center of Lancaster County, 427 N. Duke St.
Auspices: Hearing Center Association
Administrative Head: Juliet G. Stephan, R.N.; Mary Alice Hunter, M.A., Advisor
Medical Supervisor: Lloyd S. Hutchison, M.D.

118. Philadelphia, Hospital of the University of Pennsylvania, 3400 Spruce St.
Auspices: University of Pennsylvania
Administrative Head: Robert J. Hunter, M.D.
Medical Supervisor: Robert J. Hunter, M.D.

119. Philadelphia, M. V. Eversden Hearing Aid Consultant, 1918 Spruce
Auspices: Private
Administrative Head: M. V. Eversden
Medical Supervisor: None

120. Philadelphia, Philadelphia Society for Better Hearing, 2019 Spruce St.
Auspices: Independent agency
Administrative Head:
Medical Supervisor: Maurice Saltzman, M.D.

121. Philadelphia, St. Christopher's Hospital Speech and Hearing Center, Lawrence and Huntingdon Sts.
Auspices: Ear, Nose and Throat Departments
Administrative Head: David Myers, M.D.
Medical Supervisor: David Myers, M.D.

122. Philadelphia, Commonwealth Speech and Hearing Center, St. Christopher's Hospital for Children
Auspices: Department of Public Instruction, Pennsylvania
Administrative Head: Milton Brutten, Ph.D.
Medical Supervisor: None

123. Philadelphia, Temple University School of Medicine, Hearing Clinic, Broad and Ontario Sts.
Auspices: Temple University
Administrative Head: Matthew S. Ersner, M.D.
Medical Supervisor: Maurice Saltzman, M.D.

124. Philadelphia, U. S. Naval Hospital Audiology Clinic, 17th St. and Pattison Ave.
Auspices: U. S. Navy

Administrative Heads: W. C. Livingood, Commander (M.C.), U.S.N.; Joseph A. Scanlon, A.B., Coordinator of Services

125. Pittsburgh, Pittsburgh Hearing Society, Granite Building, 313 Sixth Ave.
Auspices: Private agency with Community Chest support
Administrative Head: Miss Phyllis McCalmont, M.A.
Medical Supervisor: Professional Advisory Committee

126. Pittsburgh, Department of Audiology, Eye and Ear Hospital, School of Medicine, University of Pittsburgh
Auspices: University of Pittsburgh School of Medicine
Administrative Head: Leo G. Doerfler, Ph.D., AH, F
Medical Supervisor: Kenneth M. Day, M.D.

127. Reading, Clinic for the Hard of Hearing, Reading Hospital
Auspices: Reading Hospital
Administrative Head: James E. Landis, M.D.
Medical Supervisor: James E. Landis, M.D.

128. Reading, Commonwealth of Pennsylvania Speech and Hearing Center, Trent and Belmont Avenues, Wyomissing, Pa.
Auspices: Pennsylvania Department of Public Instruction, Division of Special Education
Administrative Head: Gladys B. Fish, M.Ed., BS
Medical Supervisor: None

129. State College, Speech and Hearing Clinic
Auspices: The Pennsylvania State University
Administrative Head: E. T. McDonald, D.Ed., AS
Medical Supervisor: T. S. Mebane, M.D.

SOUTH CAROLINA

130. Greenville, Greenville Hearing and Speech Center, c/o General Hospital
Auspices: Junior League, American Hearing Society and Local Membership
Administrative Head: Oliver L. Welsh, M.A., BSP
Medical Supervisors: Paul P. Hearn, M.D.; A. J. Goforth Jr., M.D.

TENNESSEE

131. Chattanooga, Chattanooga-Hamilton County Speech and Hearing Center, 605 Vine St.
Auspices: Nonprofit corporation
Administrative Head: J. Dale Welsch, Ph.D., Director, AS
Medical Supervisor: J. E. Johnson, M.D.

132. Memphis, Memphis Speech and Hearing Center, Inc., 874 Monroe Ave.
Auspices: University of Tennessee College of Medicine, Memphis Junior League, Local Easter Seal Agency
Administrative Head: John A. Irwin, M.A., Director
Medical Supervisor: James G. Hughes, M.D., Consultant

133. Nashville, The Bill Wilkerson Hearing and Speech Center, 2109 Garland Ave., or Vanderbilt University Hospital
Auspices: Nonprofit corporation
Administrative Head: Freeman McConnell, Ph.D., AH
Medical Supervisor: Amos Christie, M.D.

TEXAS

134. Austin, University of Texas Speech and Hearing Center, University of Texas
Auspices: Department of Speech, University of Texas
Administrative Head: Jesse J. Villarreal, Ph.D., Director, AS
Medical Supervisor: Douglas Barkley, M.D.

135. Denton, Texas State College for Women Speech and Hearing Clinic, Box 3775, TSCW Station
Auspices: Texas State College for Women
Administrative Head: Kennon H. Shank, Ph.D., AS
Medical Supervisor: None

136. Galveston, Audiology and Speech Clinic, 105 Kieller Building, The University of Texas Medical Branch
Auspices: The University of Texas Medical Branch
Administrative Head: C. Olaf Haug, M.A., BH
Medical Supervisor: Fred R. Guilford, M.D.

137. Houston, Houston Speech and Hearing Center, Texas Medical Center, 6504 Bertner Ave.
Auspices: Nonprofit corporation
Administrative Head: Jack L. Bangs, Ph.D., AS
Medical Supervisor: Eye, Ear, Nose and Throat Division of the Harris County Medical Society

UTAH

138. Salt Lake City, Speech and Hearing Center, University of Utah
Auspices: University of Utah
Administrative Head: Wallace A. Goates, Ph.D., AH
Medical Supervisor: David Dolowitz, M.D.

VERMONT

139. Rutland, The Children's Rehabilitation Center, 88 Park St.
Auspices: Vermont Association for Crippled, Inc.
Administrative Head:
Medical Supervisor: Ralph H. Seeley Jr., M.D.

VIRGINIA

140. Charlottesville, Speech and Hearing Center, University of Virginia
Auspices: University of Virginia
Administrative Head: James M. Mullendore, Ph.D., AS
Medical Supervisor: Fletcher D. Woodward, M.D.

141. Richmond, Hearing and Speech Center, Medical College of Virginia, 1200 E. Broad St., Box 25
Auspices: Medical College of Virginia and Junior League of Richmond
Administrative Head: Peter N. Pastore, M.D., M.S. (A.R.L.), A.B., FACS
Medical Supervisor: Peter N. Pastore, M.D.

WASHINGTON

142. Seattle, Seattle Hearing and Speech Center, 1229 10th Ave., North
Auspices: Seattle, King County Community Chest
Administrative Head: Frederick J. Artz, B.A., Executive Director, BH
Medical Supervisor: John Tolan, M.D., Consultant

143. Seattle, Speech and Hearing Clinic, University of Washington
Auspices: University of Washington
Administrative Head: James Carrell, Ph.D. F, AS
Medical Supervisor: None

WISCONSIN

144. Madison, University of Wisconsin Speech and Hearing Clinic, Bascom Hall
Auspices: University of Wisconsin
Administrative Head: John V. Irwin, Ph.D., Director, F, AS
Medical Supervisors: Otologists serve as consultants

145. Milwaukee, Speech Clinic and Hearing Laboratory, Marquette University, 625 N. 15th St.
Auspices: Marquette University
Administrative Head: Alfred J. Sokolnicki, M.A., AS
Medical Supervisor: None

146. Milwaukee, Wisconsin State College Hearing Clinic, 3203 N. Downer Ave.
Auspices: State of Wisconsin and private support
Administrative Head: Alice Streng, M.A., Director of Exceptional Division, AH
Medical Supervisor: Thomas L. Tolan, M.D.

REFERENCE

1. Pattee, G. L., and Downs, M. P.: A Survey of Hearing Centers in the United States, A. M. A. Arch. Otolaryng. **59**:413-430 (April) 1954.

Clinical Notes, New Instruments and Techniques

A GASTROSTOMY TUBE

J. A. PERRONE, M.D., Pittsburgh

A gastrostomy tube (Fig. 1) was constructed with the idea of efficiency, more comfort, and less expense to the patient who must rely on this method of feeding for a prolonged period. It is made of soft rubber with both a distal and a lateral perforation. The proximal end has a circular piece of rubber wider than the tube itself, which gives close approximation to the abdominal wall to avoid leakage of gastric secretions and feedings. The opening of the tube is large enough to allow the end of a feeding funnel to fit with ease. A screw cap keeps the tube closed between feedings. The adult size is No. 40 F. Children's sizes are according to the size desired for the individual case.

This tube is only used after the gastrostomy fistula is formed. After the actual operating procedure a No. 40 F. catheter is used in order to get a good-sized tract. The advantage of a No. 40 F. gastrostomy tube is that thick liquids can be given with ease.

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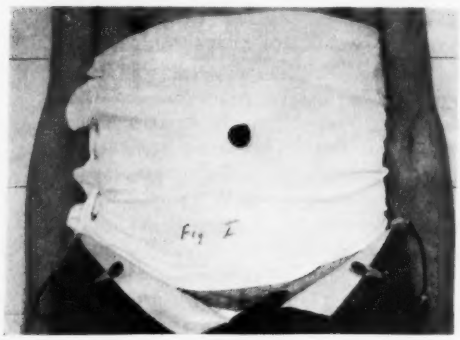
Fig. 1.—Gastrostomy tube in position.



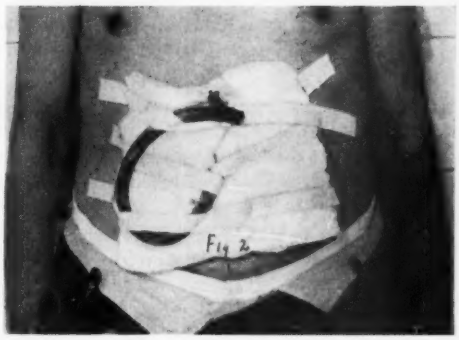


Fig. 2.—Ordinary tube with dressings to hold it in position.

I have found that this tube eliminates "bulging," which was characteristic with the old method (Fig. 2). The patient is able to dress with ease without the abdominal protrusion of the ordinary tube with associated dressings. It eliminates the use of adhesive tape and gauze completely. Patients have found it much easier to feed themselves due to the fact that the proximal end of the tube is short and well anchored. All that is needed is an abdominal binder with a perforation to admit the screw cap. Lately I have found that, instead of an abdominal binder, a two-way elastic belt with a zipper on the side, in place of the pins, has been very satisfactory. This tube was made by the George P. Pilling and Sons Company, Philadelphia.

Mercy Hospital.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Norman Leshin, M.D., *President*

Stanton A. Friedberg, M.D., *Secretary*

Monthly Meeting, March 7, 1955

An Audiologic and Otorhinologic Evaluation of Cleft-Palate Children. DR. MURRAY M. HALFOND, Temple University, Philadelphia (by invitation), and DR. JOHN BALLENGER.

In an attempt to learn more about the relationship of cleft-lip and cleft-palate hearing loss, 69 cleft-lip and cleft-palate persons between the ages of 8 and 21 years were examined audiologically and otorhinologically. The cases were divided into two groups on the basis of the presence or absence of hearing loss. It was found, however, that the group of cleft-lip and cleft-palate cases without hearing loss had minor auditory deficiencies that precluded labeling it a "normal hearing" group.

Further audiologic analysis revealed that the group with hearing losses had mild to moderate conductive-type losses, with perceptive-type elements in some ears. Analysis of the audiometric contours of the conductive components of the hearing losses showed five basic patterns could be evolved. Whether these conductive component patterns were typical of this group, all cleft-lip and cleft-palate persons with hearing loss, or persons with conductive-type hearing losses could not be determined. The conductive component audiometric patterns in the group without hearing losses were flat but showed similar contours of less magnitude than those of the hearing-loss group. In both groups, differences between right and left ears were significant.

The otorhinologic evaluation indicated that active middle-ear pathology, eardrum scarring, tonsillar pathology (both past and present), small Eustachian orifices, and abnormal tubal patency were related to hearing loss in these cases. Variables such as middle-ear fluid, eardrum perforation, adenoid pathology, velar and pharyngeal mobility, oversized turbinates, deviated septa, and frequency of upper respiratory infection were not related to hearing loss. However, when the combined frequencies of structural and pathologic stress to hearing were compared, a significant difference was found between the two groups. Therefore, it appeared that some variables contributed to the hearing loss in some cases and not in others. It was apparent in the study that the differences between groups were differences in degree rather than in kind.

Results of this study suggested that the hearing problems of the cleft-palate and cleft-lip persons need more extensive attention clinically. For the speech therapist and audiologist, the implications were that these hearing losses would not interfere with speech development but might interfere with the precison of speech production. Speech therapy methods must necessarily be adapted to the hearing loss with greater use of visual, kinesthetic, and tactile training methods. The severity of hearing losses also indicates the need for speechreading in the regular class room. Hearing aids, auditory training, and special schools would not be required.

The implications from this study for plastic surgery are the need for greater care of the cleft-lip and the cleft-palate patients, in order to reduce to a minimum all structural abnormalities, and further study of surgical technique, to insure less damage to structures related to the ear. The need for inclusion of the otologist on the cleft-palate rehabilitation team is important. In that way, all auditory deficiencies and/or otorhinologic pathology can be investigated in order to forestall or limit hearing loss.

This study served to point out the need for more careful consideration of the audiologic-otorhinologic problems that are often related to the cleft-lip and cleft-palate handicap, in order to secure more adequate rehabilitation.

DISCUSSION

DR. JOHN BALLENGER: It would appear to me that the next step in an investigation of this sort would be concerned with the function of the Eustachian tube. In man, the Eustachian tube has no good functional test. The pneumophone designed by Van Dishoeck is helpful but not

entirely reliable. Use of this was made in this study, although the report has not been given in the abstract as presented. We need a Eustachian tube function test that is reliable, and if we had that we could control the findings both in cleft palate and in other ear disease. It is hoped that further research will provide more reliable tests.

DR. EMANUEL SKOLNIK: It is gratifying to know that Dr. Halfond's findings were similar to those we have found at the University of Illinois. About 45% or 50% of the children did have hearing abnormalities. We have been convinced that the adenoid has an important function. We have seen many cases that have been repaired, and when the adenoids were removed the speech defect was greater. I am wondering if you are concerned as we are about adenoid tissue, and I wonder if you would elaborate on your findings relative to adenoid tissue.

DR. MURRAY M. HALFOND: With reference to removal of adenoids in cleft-palate cases as a threat to hearing, this is recommended on the basis of my observation and clinical judgment. Where a child has pathology, removal may be advisable. So far as the removal of lymphoid tissue about the Eustachian tube is concerned, I do not feel qualified to say whether or not that might be best to do.

The nature of the examination was of great concern to us, as was how we were going to measure deviations and how to treat the information we obtained. After careful consideration we knew of no better way to handle these examinations. Out of this we established a research approach in which we would consider only gross deviations, ignoring small deviations that might have no bearing on our cases. I would certainly grant that there were limitations on our findings, but most of the recommendations suggested, I think, have some application. This we hoped would stimulate more research and, with more study of cleft-palate cases, we might have a better understanding of the problem.

Orbital Mucoceles. DR. RANS VON LEDEN.

Orbital mucoceles originate in the frontal or, occasionally, in the ethmoid sinuses. Obstruction of the nasofrontal duct (or of an ethmoid cell) is followed by inflammatory changes in the mucous lining of the affected sinus and collection of mucus in the obstructed cavity.

The increasing pressure gradually erodes the bony orbital wall, with herniation of the cystic tumor into the orbit. Because of their insidious development, mucoceles often remain undetected until the orbital mass displaces the eyeball, causing visual changes.

The diagnosis of an orbital mucocele is based upon (1) a thorough E. N. T. examination which shows a firm swelling along the superior orbital rim and usually also obstruction of the nasofrontal duct; (2) complete eye studies which generally indicate proptosis of the involved eyeball, limited motion of one or more eye muscles, and diplopia with or without impairment of visual acuity, and (3) roentgenograms revealing cloudiness of the involved sinuses and destruction of the bony orbital roof.

Treatment consists of careful surgical removal of the diseased structures by an external approach and restoration of a patent nasofrontal passage. This may be maintained by the insertion of a large tantalum prosthesis, which can safely be left in place for several months without any discomfort.

The differential diagnosis includes all neoplasms of the orbit. The latter show no clinical signs of sinusitis, no roentgenographic evidence of sinus disease, and no destruction of the bony orbital roof. Surgical exploration confirms the diagnosis of an orbital tumor and permits its complete removal.

Early diagnosis of orbital mucoceles and prompt indicated surgery avoid the permanent impairment of vision and the grotesque appearance commonly observed in late cases.

Clinical Application of Automatic Audiometry. DR. RAYMOND CARHART.

Now that automatic audiometers are becoming commercially available at moderate cost, the clinician must become aware that the methodology employed in automatic audiometry can have a substantial effect on the test results. On would expect that some patients would find it difficult to manipulate an automatic audiometer. More basic is the fact that the level at which even a normal hearer traces his threshold can be modified significantly by the nature of the stimulus. The span between the peaks and valleys of the tracing can be sharply reduced by slowing up the rate at which the stimulus intensity is allowed to fluctuate. Again, the threshold for a tone which is interrupted by rest periods of about a second can be 10 db. better than the threshold

for the same tone when presented continuously. Finally, the normal adaptation of the ear to a previous listening situation may modify the thresholds for continuous tones by another 10 db. These factors indicate the need for a standardized technique of automatic audiometry which will reduce those variables that can be controlled and which will furnish clinicians with a firm basis for exchanging findings.

DISCUSSION

DR. ROBERT HENNER: I should like to express appreciation for this very timely discussion on automatic audiometry. I should like to know if Dr. Carhart has had any experience with differential diagnosis between conduction hearing loss and nerve deafness. What little experience I have had gives me the impression that the conduction deafness threshold is more difficult to establish than the threshold for perceptive deafness. It might be that with mixed deafness it would be possible to define the degree of nerve involvement by the breadth of the tracing.

DR. RAYMOND CARHART: We have not attempted to use automatic audiometry in clinical work. We have used it only for research. Therefore I can answer only from what is in the literature. There are many people, from Europe particularly, who claim that the spikes are much larger when the lesion is neural than those obtained if the lesion is of cochlear origin. They feel that differential diagnosis can be made on that basis. However, it is true that there are persons with completely normal hearing who have spikes so small that they would have to be classified as having cochlear pathology if the aforementioned principle were invoked. Thus, it seems to some of us that the whole question needs additional clarification through research.

Relation Between Size of Fenestra and Hearing Improvement in Fenestration Surgery.

DR. GEORGE E. SHAMBAUGH JR. and MR. JORGE GARCIA GOMEZ.

The possible relationship between size of fenestra and resultant hearing was investigated by Rytzner at Northwestern University in 1954 by means of pure-tone hearing tests at various stages of the fenestration operation. This well-controlled and careful study revealed no significant improvement in hearing during the fenestration operation until the beginning of the actual fenestra opening. At the first tiny fracture of the last layer of bone the first small gain in hearing occurs, averaging 2.1 db. for the speech frequencies. With removal of the endosteum from this opening there is a slight further gain. As the fenestra is enlarged to full size with removal of the endosteum the hearing improves considerably, to 12.26 db. above the preoperative level. Since these measurements were made with the fenestra and middle-ear opening not yet covered by the flap, they are not comparable to the situation in the healed fenestrated ear.

To investigate the possible influence of fenestral size on hearing improvement in the healed ear, 311 consecutive cases of ideal suitability were studied, operated on by two surgeons. The size of the completed fenestra, measured at operation, varied from 2 to 3.6 mm.^{2}, the width varying from 0.75 to 1.2 mm., as measured with the fenestrometer. Four groups of cases were studied: 100 operated on by surgeon A, tested six months after operation; 57 operated on by surgeon B, tested six months after operation; 100 operated on by surgeon A, tested two years after operation, and 54 operated on by surgeon B, tested two years after operation. In all four groups the smaller-size fenestras yielded a slightly lower percentage of cases at the 30 db practical level than the medium and large fenestras. Surgeon B, whose fenestras averaged somewhat larger than those of surgeon A, had a slightly higher percentage of cases reaching the practical level.

The failures in this series of 311 cases were studied. No consistent relationship was found between size of fenestra and incidence of closures, which totaled six, or in the failures due to serous labyrinthitis, which totaled three. To further invesigate the possible relationship between size of fenestra and degree of postoperative labyrinthitis, the two-week hearing tests and the two-week tests for the 4096 frequency alone were studied and no consistent difference was found in the average hearing gain or loss for the different sizes of fenestras.

In conclusion, the authors suggest that there is a very definite relationship between size of fenestra and hearing result. The optimum size seems to be about 1 by 3 mm., with a total area of 3 mm.^{2}, which approximates the average size of the oval window.

DISCUSSION

DR. ROBERT HENNER: I have enjoyed this paper, and, as a surgeon practicing fenestration, I have given some thought to the problem and think it important to have had these results presented at this time. It would seem extremely difficult to predict what the ultimate size of

the fenestra would be, because in every operation some closure must occur. I would say that the larger the fenestra the better. It is impossible to widen it more than 2.5 to 3 mm. without causing projection of the membranous canal. Physically, it would seem that the larger fenestra would have more capability of transmitting tones of lower frequency. These results were all of higher frequencies than would be affected by smaller fenestras, and therefore it is difficult to compare the results.

DR. GEORGE E. SHAMBAUGH JR.: Dr. Henner has made two excellent points: first, that a certain number of fenestrations may partially close, resulting in narrowing of the fistula. Since these patients maintain a good hearing improvement and are not revised, there is no way of knowing how many fenestras which are made of what we consider the ideal size subsequently become smaller.

The second point concerns a study of the improvement of the lower frequencies in relation to the size of the fenestra. We have observed that when a fenestra closes it is the lower frequencies that decline first. It is quite possible that the improvement for the lower frequencies will show greater differences according to the size of the fenestra than that for the speech frequencies which were considered in this study.

Norman Leshin, M.D., *President*

Stanton A. Friedberg, M.D., *Secretary*

Monthly Meeting, April 4, 1955

Simplified Techniques of Direct Laryngoscopy. DR. ROBERT B. LEWY.

This paper represents a progress report on the use of the detachable gear powered laryngoscope holder. It also describes some changes in anesthesia and position techniques and presents a new method for the open-field method of peroral laryngoscopy.

The experience of an estimated 1000 cases done with over 100 gear power standard tubular laryngoscope holders has been almost uniformly satisfactory and has made it easier for the operators to expose the anterior commissure, to demonstrate and carefully examine the larynx, and to remove polypoid and other growths with craftsman-like ease. The position of putting four sheets under the patient's interscapular region and placing the vertex of the head on the table has worked well. It has eliminated both the head holder and the shoulder holder from the operating team, a desirable thing in the average community hospital.

Using 100 mg. of meperidine (Demerol) intravenously, given in three minutes by the clock, has been advantageous. The addition of 50 mg. of meperidine to disturbed and apprehensive patients, where the dose was inadequate before, has been useful as well.

For an open-field method of peroral laryngoscopy Lewy presented a new tandem system of instruments, much lighter and simpler to use than the standard Lynch apparatus. This is composed of (1) the gear power laryngoscope holder integrated with (2) the standard Jackson slide laryngoscope, in turn integrated with (3) a slightly modified Jennings mouth gag. The laryngoscope holds up the larynx, the mouth gag holds the mouth open, and removing the slide of the laryngoscope gives an open field. Local anesthesia is possible for this work, but general anesthesia is recommended.

DISCUSSION

DR. BURTON SOBOROFF: This brief presentation by Dr. Lewy is deserving of discussion. We have used his instrument on a number of cases and found it a worth-while, handy instrument. However, there is a question as to whether or not there is need for such an instrument and whether this one meets the needs that may be present. We feel that in expert hands such mechanical assistance may not be necessary. However, in doing direct laryngoscopy work this instrument does provide very definite help. It is much like power brakes on the newer cars. Cars used to stop very well, but the addition of power brakes made them much more easily handled.

We have found this instrument worth while from three standpoints; first, from the patient's viewpoint, it has been simple to apply. I have not had an opportunity to use other similar instruments, but I imagine they could be applied as well. Also, from the viewpoint of the operator, it permits him to insert the scope and leave it there without strain, which is marked in many instances. It permits him to operate slowly and carefully in work on the vocal cords.

Whether it will avoid other operative procedures I am not certain, but I am sure it will permit careful work, a factor which could not be obtained otherwise in many cases, and, finally, it has a definite use for teaching purposes. It permits one to insert the laryngoscope without strain and without the possibility of movement if one steps aside to permit visualization of the larynx by someone else.

DR. HARKISHEN SINGH: I should like to ask in how many patients the so-called modified suspension apparatus has been successful; secondly, in how many cases the mandibular block has been successful, if any.

DR. ROBERT B. LEWY: I want to thank the discussors very much indeed. I am glad that the gear power tubular laryngoscope system described has worked so well for Dr. Soboroff. It is my feeling that the ultimate test of any newer system or idea stands or falls more on the experience of the ultimate users than on that of the originators. All work must of necessity be subjected to this discipline for final determination of its value.

Other than in the first part of the paper, this was not intended to be a statistical report but rather a presentation of some ideas and work, part of limited value and part a dissection and analysis of the mechanics of the open-field problem with work performed. In this group I have had about two sucessful mandibular blocks, but, as indicated in the paper, I found the foramen ovale more easily sought after than found, and this was not too satisfactory. My recommendation then goes toward general anesthesia rather than a nerve block method. I have had a few reasonable successes for the system of open-field work presented. These followed a series of unsatisfactory cases in which I attempted the same thing, using a clamp to join the laryngoscope to the mouth gag. I hope that the technique in other hands may bear out my own optimism concerning this system.

Restoration of Hearing in Otosclerosis by Mobilization of the Fixed Stapedial Footplate: An Analysis of Results. DR. SAMUEL ROSEN* and DR. M. BERGMAN,† New York (by invitation).

Ideally, a surgical procedure for otosclerotic deafness should reestablish the normal function of nature's pathway of sound conduction, i. e., (1) an intact and freely mobile ossicular chain terminating in (2) a freely movable stapedial footplate in (3) nature's oval window. The technique presented here was therefore devised in the belief that hearing would be restored if the fixed stapedial footplate could once again be rendered freely movable.

From 1876 to 1900 various attempts at mobilization of the stapes seem to have failed and were therefore abandoned in favor of the present-day fenestration operation of Lempert. These operations on the stapes were probably discontinued because they were performed through a myringotomy opening, which in many cases did not allow sufficient vision for safe and precise manipulation of the stapes.

The present technique is performed under local anesthesia through an ordinary ear speculum in the external auditory canal. After the drum is lifted out of its sulcus, a portion of the bony canal wall is removed in order to have under full view the vital structures to be manipulated. The operation appears deceptively simple and therefore should be practiced for a long time on the cadaver.

The results in 211 cases operated on between April, 1952, and August, 1954, are statistically analyzed.

In Class A cases (maximum preoperative loss of 10 db. by bone conduction in the speech frequencies) 52% achieved pure-tone levels of hearing between normal and 35 db. and 64% reached these levels by speech audiometry. In Class A cases 48% reached the 30 db. level by both pure tones and speech audiometry. In this group two patients achieved hearing within normal limits in the speech range.

In Class B cases (maximum preoperative loss of 20 db. by bone conduction in the speech frequencies) 39.3% achieved pure-tone levels of hearing within normal and 35 db. and 41.4% reached these levels by speech audiometry. In this class 32% reached a pure-tone level between normal and 30 db., while 34% reached these levels by speech audiometry. In this group five patients achieved hearing within normal limits in the speech range.

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In Class C patients (maximum preoperative loss of 30 db. by bone conduction in the speech frequencies) 26.6% achieved pure-tone levels of hearing between normal and 35 db. and 28.5% reached these levels by speech audiometry. In this category 16% reached the 30 db. level or better by pure tones, while 21% reached this level by speech audiometry.

When percentages of improved patients in the categories most suitable for mobilization (A and B) are combined in accordance with the number in each category, 42% reached pure-tone levels between normal and 35 db. (46% by speech audiometry), while 36% achieved a hearing level between normal and 30 db. If the percentage of improved patients in the Class C category (generally less suitable) are added to those of A and B, 37% reached levels of 35 db. or better by pure tones (40% by speech audiometry), while 30% reached a level of 30 db. or better by pure tones (32% by speech).

A total of seven patients obtained a postoperative hearing level within normal limits (less than 10 db. loss).

In none of the unsuccessful cases did the hearing fall significantly below the preoperative level.

Patients with early otosclerosis, with a hearing loss of 30 db. or less, may be ideal for mobilization, since the hearing loss is not great enough for fenestration. Successful mobilization may also contribute to the auditory rehabilitation of a patient with severe otosclerotc deafness when the conductive element is reduced or eliminated.

A case is presented showing that mobilization of the fixed stapedial footplate restored hearing in an ear previously fenestrated without success.

A patient with otosclerotic deafness now has two opportunities for surgical restoration of hearing: (1) mobilization of the fixed stapedial footplate and (2) fenestration.

DISCUSSION

DR. GEORGE E. SHAMBAUGH JR.: This has been a very interesting presentation. Of one thing I am sure: This procedure deserves a fair trial before pronouncing judgment for or against it. Dr. Rosen is to be congratulated on his pioneering in this field. As he himself mentioned, he is not the first to attempt mobilization of the stapes. The fact that it had been tried before and abandoned does not necessarily mean that with improved technique it may not give satisfactory results.

Regarding the permanency of improvement, time alone will tell. Logically we would expect the otosclerotic lesion to cause reankylosis. However, in following a large number of fenestration operations we have observed that the otosclerotic lesion frequently remains quiescent for a period of years, and it is possible that such a quiescent lesion might not reankylose at least for a considerable period of time.

The indications for the mobilization procedure still need to be more clearly defined. Dr. Rosen apparently includes all cases with stapes fixation as candidates for mobilization. With this I would not agree, at least until further studies have indicated that mobilization can yield as good results in the ideal case as fenestration. At the present time, we are reserving for mobilization the cases which are not yet sufficiently fixed to warrant fenestration and cases with considerable cochlear degeneration, making them of questionable suitability for fenestration.

A very intriguing possibility of the mobilization is the fact that theoretically we might restore normal hearing. We know that fenestration does not restore normal hearing, leaving an average conductive residue of about 25 db. for the speech frequencies. Theoretically, with an intact ossicular chain a completely mobilized stapes might restore the hearing to normal without a conductive deficit. I have had the opportunity to observe Dr. Rosen's work and of testing audiometrically several of his patients who were successfully operated on. One of these patients had had a fenestration done elsewhere, with an excellent result. Subsequently Dr. Rosen performed his mobilization procedure on the opposite ear, and there was no question but that the mobilized ear had the better result. However, while it is theoretically possible for mobilization to restore the hearing to normal, it is disappointing that the majority of Dr. Rosen's operations have shown a residual conductive loss approximating a successful fenestration.

Our own experience with this procedure is limited to 15 operations, with only 3 that have shown a maintained hearing improvement as good as, or better than, might have been obtained by fenestration. Two of these patients with considerable cochlear nerve degeneration received a hearing result from mobilization exceeding the predicted level from fenestration. Against these

few encouraging results are several cases with a further drop in hearing in the first test one week after operation and one patient whose stapes was mobilized as part of a fenestration operation, resulting in bleeding into the labyrinth, probably from luxation of the stapes footplate. This experience indicates the possibility of possible cochlear damage from an attempt at stapes mobilization. In five patients with unsuccessful mobilization, fenestration was subsequently carried out, with excellent results in all five. However, although an unsuccessful mobilization may later be successfully fenestrated, patients who have once undergone unsuccessful surgery may be reluctant to undergo a second operation.

In closing, I should like to mention an interesting observation for what it may be worth. A patient came to us with a bilateral hearing loss due to otosclerosis with a large amount of cochlear involvement and only slight stapes fixation. Particularly annoying to her was a roaring tinnitus in one ear. According to the prediction formula, fenestration offered no possibility of improvement. Mobilization was suggested and eagerly accepted in the hope that the terrific tinnitus might be lessened. In exposing the stapedial tendon, the chorda tympani nerve was cut and immediately the patient mentioned that the terrific roaring in her ear had ceased. This was before the stapes had been mobilized. She has been followed for a month since operation, with no recurrence of the roaring tinnitus. She also shows no hearing improvement in this ear. I must say that it is difficult to explain this observation, since there is no good scientific evidence that the chorda tympani is related to hearing.

DR. CLAIR M. KOS, Iowa City: It is a great pleasure to discuss the subject of stapes mobilization as presented by Dr. Rosen and Dr. Shambaugh. It is with some regret that my remarks must be made without having had the good fortune to observe Dr. Rosen's technique or know what he does, except from his publications. In many respects the data presented by Dr. Rosen and Dr. Shambaugh converge to a point of agreement with mine. Dr. Rosen has said nothing with which I can significantly disagree.

My experience has been limited to 65 stapes mobilization operations, and from this the following significant facts and impressions have been revealed: Diagnosis must be a hit-or-miss procedure; there are no detailed criteria evident for selection. Prediction cannot be made with a significant degree of confidence. It is apparent that at least three categories of clinical otosclerosis must be considered for application of mobilization. The first one is based on normal bone conduction for pure-tone audiometry, with a range of from 20 to 40 db. for the air conduction thresholds. The second category includes complete stapes ankylosis in which the air conduction threshold exceeds the normal bone conduction threshold by 40 db. or more; the third, stapial ankylosis of any degree with inner-ear ankylosis. Regression occurs in the first and second groups but more frequently in beginning ankylosis; within four to six weeks postoperatively it is most frequent. It has occurred six months postoperatively in this series of patients.

The operation seems to have greater effectiveness in the third group, in which bone conduction thresholds are elevated to such an extent as to preclude the fenestration operation. The technique employed does not compromise subsequent fenestration. After three to six months there is nothing to alter the effectiveness of fenestration. Complications are negligible. Postoperative bleeding occurred in three cases. One patient had serous labyrinthitis; this occurred before antibacterial coverage was used. Penicillin is given intramuscularly the day before and the day of operation. The hearing may be made worse in beginning anklyosis in which the crura are fractured. The usual loss is not more than 10 db.

Of the total of 65 patients, 20 received serviceable hearing or to a level of 30 db. or above. If this is broken down, which I shall not take the time to do because the statistics vary little from those of Drs. Rosen and Shambaugh, it is apparent that stapes mobilization is not a reasonable substitute for fenestration, as its application is not sufficiently effective in cases suitable for fenestration. In incomplete ankylosis 80% were improved, but in the firmly fixed cases only 20% had improvement. This is impressive, but it is not sufficient to employ it in all cases in which the stapes is completely fixed. It seems to be useful in beginning ankylosis and where bone conduction is elevated to a point that precludes successful fenestration.

(Movie showing technique of operation.)

DR. JOHN R. LINDSAY: We must congratulate Drs. Rosen, Shambaugh, and Kos for giving an excellent presentation of this new procedure. So far no one has shown any microscopic slides to indicate just what was required of this operation or what may have been accomplished

by it. If there is bony fixation of the stapes and you break it, the hearing does not return to normal except in a relatively few cases, nor does it improve to the same degree in all. Another point, what is the pathologic picture in far-advanced otosclerosis, and what can be expected of attempted mobilization?

I show a section from an early case of otosclerosis to illustrate the condition around the footplate of the stapes. There is a great deal of fibrous thickening around the footplate and an irregular formation of bone. When there is early ankylosis in a case like this, and it is broken loose, a return to normal function could hardly be expected because of the fibrosis about the joint. I think this may be one reason why one may not get a spectacular improvement even in early cases.

If we look at some pathologic slides from advanced cases of stapes fixation we see that it is not just bony ankylosis at the joint, but an extensive change in the whole stapes footplate and crura. It seems obvious that to break such a stapes loose would be most difficult or impossible.

In the next slide from a far-advanced case of otosclerosis one can see a similar very advanced and extensive change. There was profound deafness. With such a change in the footplate of the stapes and at the joint, it would be practically impossible to break it loose.

My experience with mobilization is limited to three cases. The first was a case in which I had done a fenestration in one ear, and later the patient's husband accidentally mobilized the stapes in the other ear by means of a bobby pin and got a better result than I did. She still has a good result after two years. I have done two cases since then by the operation of Rosen. One was a failure; the other was successful. I do not wish to discourage the procedure. I notice that Dr. Rosen included cases with profound otosclerosis and deafness as one category of cases for operation. The extent of the pathologic change would probably preclude the possibility of doing a successful mobilization in most of the advanced cases. The nature of the pathologic lesion in the particular case probably has much to do with the chance of success.

DR. M. BERGMAN: I have been closely associated with some of the statistics and am impressed with several aspects of the work. As one interested in rehabilitation, I find one point in particular which I think important. This is concerned with the case where there is a profound loss of hearing due to otosclerosis, where bone conduction is down, where air conduction has reached the level of 70 to 90 db., and where closing the gap can mean a considerable difference to the patient. We know the limitations of a hearing aid; a patient with a 90 db. loss cannot be helped in this manner very much. We have cases that have shown 20 to 30 db. improvement following stapes mobilization, not enough to close the gap completely, but this gain made a sub tantial difference in the patient's ability to use a hearing aid. Rehabilitation with a hearing aid and lipreading would not have been as effective as that.

I want to show one slide showing improvement in hearing for 98 of the 211 cases. The question of practical hearing is not involved, since this shows the number of patients who achieved gain in hearing following successful mobilization of the stapes. The percentage is a little higher than previously shown for those whose hearing reached the "practical" level. In the Class D group (38 cases) 42% had definite improvement in hearing which may be considered an aid to rehabilitation.

I should like also to point out the close correlation between the results of the speech audiometry and pure tones in the data presented in Dr. Rosen's paper. Except for the "A" category cases, where there was a larger percentage of "successful" cases shown by speech audiometry results, the pure-tone and speech-test results seem to substantiate each other.

DR. SAMUEL ROSEN: I should like to thank Drs. Shambaugh, Kos, Lindsay, and Bergman for their discussion. My experience with the first case operated on three years ago was similar to the first case Dr. Kos did recently. In my patient the hearing is still normal on the side of mobilization. From the beginning I expected that the footplate would reankylose, but this does not seem to happen, for in all the patients who reached the 30 db. level or better after mobilization only two receded to the preoperative level.

Dr. Shambaugh showed several slides of a case in which the hearing was worse a week after he performed the mobilization operation. This finding is present after all mobilizations for about two or three weeks and is due to blood in the tympanum and reaction of the soft tissues and not to labyrinthitis, of which there are no signs or symptoms. In the 500 cases upon which I have operated I have not had a single case of labyrinthitis. In one case which Dr. Shambaugh fenestrated a week or so after mobilization it is quite possible that if he had delayed the fenestration the hearing might have improved.

The predictability of the degree of fixation of the footplate of the stapes in otosclerosis based on audiological findings will have to be revised. I do not believe it is possible to state with any degree of certainty what the degree of fixation of any particular stapes is going to be. In 500 cases in which I have used my technique for mobilization I have found that the air-bone gap is not necessarily closely related to any predictable finding in the degree of stapes fixation.

Dr. Shambaugh thinks that mobilization should be done only in those patients whose hearing has not gone below the 30 db. level and also in those with a mixed type of deafness unsuitable for fenestration, and that the large group of patients suitable for fenestration should not have the mobilization operation. I would take issue with this and include those cases for mobilization who are suitable for fenestration, because I was able successfully to improve the hearing of this group to the 30 db. level in 48% of the cases. Surely nothing is to be lost by doing the mobilization operation first. The case of which Dr. Shambaugh told us, with intractable tinnitus which stopped immediately after section of the chorda tympani nerve, tends to give us a further clue that the chorda tympani is not only concerned with taste but also with sound and equilibrium. Dr. Kos reported gratifying results in 60 cases in which mobilization of the stapes was done. Dr. Kos, as well as Prof. Y. Meurman, whom I visited in Helsinki nine months ago, was able to work out for himself my technique for mobilization of the stapes.

In discussing some aspects of this work with Dr. Lindsay this evening, it seems that we have both had the experience that, even though the footplate of the stapes seems in some cases to be moving, the mobility does not seem to be free enough. Dr. Lindsay suggests that at times a fibrosis around the footplate of the stapes prevents the hearing from attaining the higher levels, because the footplate is not rendered mobile enough.

Abstracts from Current Literature

Ear

ENDAURAL APPROACH IN TEMPORAL BONE SUGERY. LUCIAN W. ALEXANDER, Eye, Ear, Nose & Throat Month. **34**:239 (April) 1955.

Temporal bone surgery has been simplified by the endaural approach in bone surgery, with improvement of the results. The historical evolution of this technique is traced to the beginning, with Lempert's search for a simplified approach for the surgical treatment of exostosis of the external auditory canal and the postauricular fistula. The endaural technique was then applied to mastoidectomies and other surgical procedures of the temporal bone and finally to the fenestration operation.

Local anesthesia is used in the endaural approach even when a general anesthetic is administered. Alexander prefers Shambaugh's modification to Lempert's original incision, as it requires only two incisions instead of three. Conservation of the skin helps to prevent postoperative atresia of the external auditory meatus.

The endaural approach allows the most direct surgical approach to the structures of the middle ear, inner ear, and mastoid. It affords excellent visibility and eliminates any necessity for blind surgical procedures in this area. There is minimal trauma to the soft parts, excellent cosmetic results, and a shortened period of hospitalization, with an earlier return to normal activities. The technique requires a detailed knowledge of the anatomy of the temporal bone, which can only be acquired by painstaking supervised dissection in the anatomic laboratory.

JENNES, Waterbury, Conn.

WHY IS YOUR PATIENT DIZZY? GORDON D. HOOPLE, New York J. Med. **55**:2161 (Aug. 1) 1955.

Dr. Hoople, of the Department of Otolaryngology, University and Memorial Hospitals in Syracuse, N. Y., gave his report before the Annual Meeting of the Medical Society of the State of New York on May 12, 1955. The paper is quite comprehensive and therefore should be read in detail, but in summary the following highlights should be kept in mind: One must take a careful detailed history, and the physical should be complete, including many neurologic tests. If the patient is "really dizzy" he must have a sense of motion. Disturbance of position in space depends upon the labyrinthine, ocular, and proprioceptive systems. Spontaneous nystagmus, present or absent, must be looked for. In every patient with vertigo one must evaluate both the cochlear and vestibular functions. When the cause is intracranial, the symptoms are rather constant and do not have the ordinary periodicity of other causes. Explosive dizziness with nausea, vomiting, tinnitus, and hearing loss usually points to peripheral pathology. Patients with such serious persistent symptoms commonly have an irreversible condition. On the other hand, minor dizziness is often temporary and reversible. We should spend more time in studying etiology rather than treatment, which must come after accurate diagnosis. Since these patients are frightened, worried, and out of focus, they should be managed with sympathy and understanding of the psychic element. The phrase "Oh you'll be all right" is not enough.

I. W. VOORHEES, New York.

SOME LIMITATIONS OF RADICAL MASTOIDECTOMY AND THE VALUE OF EARLY CONSERVATIVE SURGERY. A. R. DINGLEY, J. Laryng. & Otol. **69**:361 (June) 1955.

A somewhat philosophical approach is taken to the question of radical mastoidectomy versus modifications of the radical mastoid operation. The subject is discussed historically, and it is shown that many of our basic precepts regarding indications for radical procedures are changing, in the light of the many well-known developments in recent years. In the same vein, the question of conservative toilet cholesteatoma of the epitympanum is discussed, and the author believes early conservative surgical treatment permits both removal of potential danger and preservation of hearing. The precepts handed down from the past may be classified under the "all or none"

law. In either case, namely, the "all" or the "none," the hearing function ultimately was lost in most cases. Present trends are to be more conservative where previously radical operation was thought to be indicated and to be more prompt to perform a conservative operation on patients that would previously have been treated by local toilet and observation. The over-all trend, when following this middle-ground approach, is to give the patient the best opportunity to maintain serviceable hearing, without placing him in danger of serious intracranial complications.

Dingley discusses his own observations of results in the modified radical operation as to subsequent hearing and relative danger. He observes that the preoperative hearing level is of most importance in determining how conservative one may be; and, generally, the better the hearing, the more conservative one may be. His best results have been obtained in attic disease, and high posterosuperior marginal cases are next. An intact tympanic membrane postoperatively preserves hearing, protects the windows of the labyrinth, and covers the Eustachian-tube orifice.

Hearing results are tabulated based upon the postoperative presence or absence of the incus. The author feels that the absence of the incus due to disease or surgical removal results in only 15 to 20 db. loss of hearing and that subsequent removal of the drum is not justified merely on the basis of absence of the incus.

A final observation is that often postoperative cavities fail to become dry in the presence of active disease in the opposite ear; eradication of disease in both ears will result in bilateral healing. Eustachian-tube spread is postulated.

LeJeune and Stevenson, New Orleans.

Primary Results of Tympanoplasty. L. Pietrantoni, E. Bocca, and C. Agazzi, Rev. laryng. **76**:10 (Jan.-Feb.) 1955.

The authors present their results as obtained in the use of grafts in radical mastoidectomies. Their findings are evaluated primarily on the basis of postoperative audiometric results. Their technique is used in both complete radical and modified radical procedures. They classify the pathology in three groups: (1) where the lesion involves only the tympanic cavity without encroaching on the ossicles, (2) where the lesion involves the tympanic cavity and only two ossicles are involved, and (3) where there is encroachment of all the ossicles.

In Group I, their technique involves the endaural approach, with the removal of the diseased pathology but the conservation of the tympanic ring. Then a full-thickness skin graft is applied which covers the epitympanum and also the perforation of the tympanic membrane. In the second group of cases the tympanic ring is obliterated and the graft is applied more extensively, but this time it covers the stapes. In the third group of cases all the ossicles are removed, including the stapes. Then the authors occasionally insert an acrylic implant, placing it over the oval window. The skin graft is inserted, covering the entire exposed and denuded cavity and also a residual perforation in the tympanic membrane.

In summarizing their results, they found that there is either a considerable preservation of hearing or marked improvement in many of their patients. They apparently are not concerned with the region of the eustachian tube, since they believe that maintenance of the patent Eustachian tube will not only maintain a better hearing level but also add to the improvement of any hearing loss.

Persky, Philadelphia.

Pharynx

Prolonged Anesthesia in Tonsillectomy. Walter E. Berman, Eye, Ear, Nose & Throat Month. **34**:181 (March) 1955.

Observations were made in the control of postoperative tonsillectomy pain by the use of Efocaine. This was injected following tonsillectomy under both general and local anesthesia in a series of 150 patients, and another 150 patients, who were not injected, served as controls. One cubic centimeter of the solution was infiltrated in the anterior and posterior pillars on each side, a total of 2 cc. for each patient. The subjective results were obtained by questioning the parents of the children and by questioning the adult patients themselves. Of the 150 injected patients 84% had little or no postoperative complaints and ate well from the first day, whereas in the control cases only 26.66% had little or no postoperative complaints. The duration of prolonged anesthesia pain was observed for approximately four to six days.

Abstractor's Comment: It is admittedly difficult at best to evaluate purely subjective results. This may explain the variations in the results obtained in the use of Efocaine. In an

attempt to eliminate many of the variables, I tried injecting a small series of patients with Efocaine, injecting only one side in each patient. The patients were unaware of the side which was injected and were questioned on the first postoperative day and at the end of seven days as to any differences of pain sensation between the two sides. It is, of course, not uncommon for patients following tonsillectomy to comment that one side hurts less than the other, and I found that in this small series that this remained true and that the side which a patient considered as hurting less bore no relationship to the side of injection. One would have expected a more consistent reference to the side of injection.

JENNES, Waterbury, Conn.

BENIGN TUMORS OF THE TONSILS. HANS BRUNNER, Eye, Ear, Nose & Throat Month. **34**:248 (April) 1955.

Benign tumors of the tonsils, although uncommon, may cause such clinical symptoms as nausea, vomiting, and choking sensation if they come in contact with the base of the tongue. Brunner cites three cases of benign tumor of the tonsil which caused symptoms. These included a squamous papilloma, a benign hyperplasia of inflammatory origin, and a cholesteatomatous cyst.

JENNES, Waterbury, Conn.

PHARYNGOESOPHAGEAL DYSKINESIA AND THE CERVICAL SYNDROME. J. TERRACOL and Y. GUERRIER, J. franç. oto-rhino-laryng. **4**:17 (Jan.) 1955.

The authors discuss the problem of a painful throat with a sense of obstruction and difficulty in swallowing. The local findings are usually minimal. If there is any evidence of an inflammatory process, it is practically negligible. Most careful examination usually fails to reveal either any local pathology or disturbance of function. The authors believe that the symptoms are of a reflex nature, due to an arthritis of the cervical vertebrae, and that treatment to this area is usually followed by considerable relief of pharyngeal symptoms.

PERSKY, Philadelphia.

Larynx

TREATMENT OF BI-LATERAL ABDUCTOR VOCAL CORD PARALYSIS. H. P. LAWSON, J. Laryng. & Otol. **69**:374 (June) 1955.

A very orderly step-by-step discussion of the over-all problem of bilateral abductor vocal cord paralysis, and evaluation of various methods of its treatment, is present. The incidence, symptoms, and disabilities are mentioned. The main body of this paper may be divided into (1) indications for surgical treatment and (2) evaluation of results obtained by the various surgical procedures employed. The indications are divided into four groups and outlined very thoroughly.

In assessment of surgical results, the author emphasizes that tracheostomy should always be thought of in terms of a temporary measure. Definitive surgical procedures mentioned include incision of the cord. Subsequent scarring and stenosis nullify the initial improvement in this procedure. A transposition of the cord through a laryngofissure is described without comment. King's operation is described, and the comment is made that attachment of the omohyoid to the arytenoid is of no value. The Kelly procedure is also described. Both the King and the Kelly procedures are criticized because of the technical difficulties and because they believe the optimum airway is obtained immediately and subsequent scarring tends to reduce the glottic aperture.

The Hoover submucous excision of the cord is described, followed by the author's modification of this technique. The author's technique is well illustrated in the article. The three basic features are the following: 1. Laryngofissure exposure. 2. Two parallel mucous membrane incisions, one 1 mm. above and 1 mm. below, and parallel with, the edge of the vocal cord. This is followed by submucous excision, under direct vision, of the muscle and arytenoid. 3. Fine closure of the mucous-membrane edges, followed by suturing of the mucous-membrane flaps to the underlying thyroid ala with mattress sutures.

The author's results have been good regarding airway but admittedly disappointing regarding voice. Postoperative voice therapy is advocated following all types of such plastic procedures on the vocal cords.

LEJEUNE and STEVENSON, New Orleans.

Nose

DISCUSSION ON THE RELATION OF NASAL DISEASE TO ASTHMA. WILLIAM BROCKBANK and KATE MAUNSELL, Proc. Roy. Soc. Med. **48**:509 (July) 1955.

The Section of Laryngology met under the presidency of Prof. V. F. Lambert, M.D., Ch.M., F.R.C.S., on Feb. 4, 1955. Dr. Brockbank spoke of a dozen years in the asthma clinic and said that he was "struck" by the number of asthmatics who have nasal symptoms. There was often anterior purulent discharge or postnasal drip, soft or bony nasal obstruction, or evidence of nasal allergy. However, the patients' histories showed 27% denial of nasal difficulties. The x-ray films showed only about 45% sinus opacity, although the percentages were not quite definite, since there was only 1 "completely opaque antrum" in 161 patients. Brockbank agrees that a hazy antrum does not always mean sinus infection, but may be simple swelling of the sinus mucosa. Sinus washings were done on 30 antra in 24 patients. Nineteen were clear, and only eight showed purulent fluid. Such results were deemed "disappointing." Some patients are relieved, especially children, by sinus irrigation plus an appropriate antibiotic. As for nasal surgery in these cases, he states: "I have only on one occasion seen any good come out of . . . operations"—septal resections, turbinectomies, etc. But a 10-year-old child was greatly relieved of asthma by removal of a bony septal spur. Brockbank wonders whether in allergic cases it were not well to "keep surgeons away from noses at all costs."

Dr. Kate Maunsell spoke on the relation of allergic rhinitis and bronchial asthma. Sir Henry Dale and Sir Thomas Lewis demonstrated that in allergy there is a liberation of histamine. This not only causes edema in the mucosa of the respiratory tract, but constriction of the smooth muscles of the bronchi. Antihistamine drugs sometimes relieved sneezing, watery discharge, and edema of the nasal mucosa, but their good effect was evanescent and did not help chronic asthma very much. Incidence of asthma with rhinitis varies greatly: 24.5% in her 271 cases. The high incidence in childhood is not easily explained, nor why boys should be more afflicted than girls. Extrinsic factors are suspected, particularly dosing with drugs which relieve only temporarily. "Aspirin may cause violent asthma, and 5 grains of aspirin may be fatal." Dust and fumes and any other particles may cause asthma. The sizes of the particles which rest on the nasal mucosa have been studied. Particles larger than 100μ are caught in the vibrissae, but smaller ones will get by and invade the lower airways, especially if obstruction of the nose causes mouth breathing. Pollen in England starts an increase in the number of observed asthmatics from April to August, decreasing thereafter. Mr. J. C. Hogg, carrying on the discussion, thought heredity plays a part, but he always studies the following causes: nasal polypi, nasal allergy, suppurative sinusitis, and deflected nasal septum. He described his experiences in each of these conditions. As for cortisone and corticotropin (ACTH), they block or neutralize antigen-antibody reaction, thus lowering the hypersensitivity. As for treatment, cauterization of sensitive spots in the nose, septum, and turbinates may be helpful. It should be light and not destructive to any great degree. Zinc ionization has an effect which may be explained by migration of hydrogen ions into the cells and tissue fluids. Diathermy may be useful in a child but requires daily dosage for eight weeks or more, relieving a stuffy nose and, therefore, aiding proper breathing. It is said to be harmless. Nasal polypi are the result, and not the cause of allergy. They may be numerous, persistent, and defy complete destruction. Ethmoidectomy may be required in very severe cases where the deep ethmoid cells are not otherwise reached. Suppurative sinusitis is not recognized by all authorities as causal in asthma, but Rackenman and Weille (1939) showed that this may come about in three ways: (1) chronic irritation of trachea and bronchi from inhaled infected secretions, (2) reflex bronchospasm from inflammation of the sinuses, and (3) bacterial sensitization and subsequent allergic reactions in the mucosa. These items are discussed and expanded too extensively for consideration here, but one patient is reported who had wheezing, dyspnea, and signs of emphysema and bronchitis. The nasal symptoms were relieved by a successful Caldwell-Luc, but three days after operation he developed severe bronchial asthma, which has persisted!

In conclusion of the Symposium, the President, Professor Lambert, said that he had always impressed upon his students that in any asthmatic condition nasal obstruction must first be corrected. In reflex nasal asthma the spell could be broken by cocainizing the nasal ganglion. The trigger reflex could be in the posterior area behind the turbinates.

ABSTRACTOR'S COMMENT: Asthma may be considered to be more of a constitutional than a local disease, but we usually think in the reverse order. In any case, the rhinologist will do

what he can to clear the upper airways by whatever means his experience points the way. There is definite hypersensitivity due to whatever causes can be found. It is well, too, to keep in mind that there may be more to any given case than meets the eye. We all have seen psychosomatic instances, but they may not have been immediately apparent at the first visit. Cooperation between rhinologist and internist can be helpful to each of them and, finally, to the patient, which is the first desideratum always.

I. W. VOORHEES, New York.

THE NASAL ASPECT OF MIGRAINE. H. G. BEDFORD RUSSELL, Proc. Roy. Soc. Med. **48**:519 (July) 1955.

This is a preliminary report on the contrast radiology of swelling of the sinus mucosa. Vascular changes in certain arteries may affect head pains. The cephalic structures supplied by the trigeminal are worthy of attention. In classical migraine relief is often found after due attention to sinuses and teeth. The sinuses are not static cavities, and contrast radiology shows that certain areas of the mucosa are "highly dynamic." This alteration can be described as "irritable." Where scotoma accompanied migraine, swelling of the sphenoid mucosa was commonly found; and other sinuses exhibited a like condition, even without scotoma. In 10 cases with headache and scotoma there was evidence of sinusitis. Allergy often precedes sinusitis. It is not to be cured surgically in most instances, but by shrinking (ephedrine hydrochloride and saline irrigations). By introducing a contrast medium into the sinuses one can show x-ray differentiation of areas of swelling and of nonswelling, especially in the sphenoids. The phenomenon resembles urticaria. Local treatment often gives immediate relief and sometimes a "cure," which may last for some years. "Sluder (1912) showed that cocaine in the sphenoid paralyzed the trigeminal." At least one cause of these intrasinal swellings is bacterial allergy.

I. W. VOORHEES, New York.

Miscellaneous

ALLERGIC REACTIONS TO ANTIBIOTICS. SHEPPARD SIEGAL, New York J. Med. **55**:2303 (Aug. 15) 1955.

Siegal reported from the Allergy Clinic of Mt. Sinai Hospital in a paper read before the Medical Society of the State of New York at the Annual Meeting in Buffalo on May 10, 1955. The choice of an antibiotic is of high importance to every practitioner of medicine. First, the one chosen may be useless in the type of infection at hand. Second, it may be dangerous because of anaphylaxis; fatalities have occurred promptly in office practice. Or, very disturbing reactions may beset the patient but with apparently full recovery. Chloramphenicol was one of the worst antibiotics when it was not so well known. It caused blood dyscrasias, although pejorative changes in the bone marrow were not demonstrated. Siegal urges proper discrimination in all antibiotic medications and offers suggestions on how to avoid dangerous and untoward symptoms. Since penicillin is the most commonly used, it is well to consider that one intensively. A history of allergy in general is suggestive but is not enough unless it is very marked, since there are relatively few persons highly sensitive to this "drug," considering its wide use. However, after chlortetracycline (Aureomycin) severe attacks of asthma have been reported. Oxytetracycline (Terramycin) has caused serum sickness and in the early days of its administration was accused of impairing both sight and hearing. Gastrointestinal disturbances are still seen to be derived from this therapy.

Reactions from penicillin are classified by Siegal under five headings: 1. The delayed type, which may give no untoward symptoms for about five days. Then we may have eruption, fever, and joint pains. 2. Accelerated and immediate reactions occurring in minutes, hours, or days, with abdominal pains, weakness, and fainting, or a complete anaphylactic picture. 3. Hyperergic reactions with severe manifestations in the skin, intestines, and vascular tissues and organs. 4. Erythematovesicular or "id-like" reactions, eczematous in type, affecting hands, feet, and groins with both papules and vesicles. 5. There is also a contact dermatitis seen in laboratory workers or others who use bare hands when working with the antibiotics. Hence ointments are less used now than formerly, especially not by oculists, who used to administer antibiotic ointments to the lids.

In summary, penicillin is the major antibiotic sensitizer. Reactions to the broad-spectrum types are not commonly allergic. These penicillin allergies seem commoner in adults than in children, males being more likely to show them than females. It is possible to desensitize the

patient so that penicillin may be given when needed. Penicillin skin tests may be deceptive and therefore inconclusive. A dosage schedule is set down in making this desensitization effort. Anaphylactic shock is still to be dreaded and must be kept in mind in all cases as a possibility. Therefore, careful history taking and skin tests must be emphasized. Methods of administration must be studied and varied according to the case at hand. Slow intramuscular injections drop by drop are advisible, and entrance into a vein must never be allowed. Epinephrine should be at hand for any emergency which may arise. One should be skilled in carrying out shock treatment and know instinctively what to do. There are two tables, three figures, and 67 references to the literature in this thoroughly comprehensive article, which will be of value to all ENT workers.

I. W. VOORHEES, New York.

FREON, A NEW AND SAFE REFRIGERANT ANESTHETIC FOR SURGICAL SKIN PLANING. JOSEPH J. ELLER, New York J. Med. **55**:2352 (Aug. 15) 1955.

Plastic surgeons are often confronted by facial blemishes which are not amenable to "the knife." Acne and smallpox pits may cause endless unhappiness. Various efforts to produce desquamation, such as scrubbing with phenol, have been only fairly successful, and painful withal. Ethyl chloride has been the chief agent in producing rigidity of the skin and transient anesthesia, but it is inflammable, explosive, irritating, and when inhaled, may be dangerous in excessive amounts. The author has been using Freon "in hundreds of cases." Chemically, this agent is dichlorotetrafluoroethane and is a product of E. I. Dupont de Nemours & Co. It is a refrigerant anesthetic and is applied before using a high-speed stone grinder (30,000 rpm). The illustration to the text shows a woman with deep left-sided facial scars and pitting before treatment. After a single dermal abrasion under Freon, the skin looks clear, with excellent cosmetic result. Eller states that this procedure is likely to be standard for use in the office of the general practitioner as well as in that of the dermatologist. One hopes that this is true without equivocation and that it is as simple to use as it seems to be.

I. W. VOORHEES, New York.

A TEN YEAR STUDY OF CARCINOMA OF THE ESOPHAGUS. D. P. SHEDD, L. G. CROWLEY, and G. E. LINDSKOG, Surg., Gynec. & Obst. **101**:55 (July) 1955.

This report concerns 180 patients admitted to the New Haven Hospital in 1940-1950 with a diagnosis of carcinoma involving the esophagus. The median age of the group was 63 years. There were 22 females (12%) and 158 males (88%).

The first symptom was dysphagia, noted in 65% of the group, with pain being the second most frequent symptom in occurrence.

A preponderance of epidermoid lesions of the esophagus was noted, except in the lower areas, where adenocarcinoma predominated.

Esophagoscopy was the most valuable diagnostic method, but there was a high degree of correlation between the x-ray and esophagoscopic diagnoses. Esophagoscopy was performed in 155 of 180 cases, and in 89% of the 155 cases the diagnosis was positive.

Among the 22 females in the series 9 had lesions in the upper esophagus, 4 in the middle, 3 in the lower, and 6 in the lower and cardia areas.

There are detailed tables with the various statistical comparisons, classifications, and results of resection. The greatest survival was in those patients who underwent resection. Of those patients upon whom resection was performed, one-third died from distant metastases. In contrast, however, where resection or x-ray therapy was not carried out, approximately one-third died of local invasion of the tumor. There was an operative mortality of 19% in those upon whom resection was done and a mortality of 7% when exploration alone was carried out.

As in other clinical studies of carcinoma of the esophagus, the authors could find no reason for optimism. The case summaries of the only three survivors in the entire group are presented. These patients all had adenocarcinoma of the lower esophagus or upper cardia. No patient with epidermoid carcinoma survived longer than 27 months; no patient with anaplastic carcinoma survived longer than 7 months.

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Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. D. S. Gorrell, 110 Medical Arts Building, Calgary, Alta.
Secretary: Dr. G. Arnold Henry, 328 Medical Arts Building, Toronto.
Place: Chateau Frontenac, Quebec, Canada. Time: June 6-7, 1956.

International Bronchoesophagological Society

President: Dr. Antonio da Costa Quinta, Lisbon, Portugal.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Fourth International Congress of Bronchoesophagology.
President of Congress: Dr. Antonio Carrascosa.

International Congress of Audiology

Place: Leyden, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Holland.

International Course in Paedo-Audiology

Place: Groningen, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Holland.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Juan Manuel Tato, Azcuenaga 235, Buenos Aires, Argentina.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Fifth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Place: San Juan, Puerto Rico. Time: April 8-12, 1956.
President of Congress: Dr. Juan H. Font, Medical Arts Building, San Juan.

Sixth International Congress of Otolaryngology.

President: Dr. Arthur W. Proetz, 3720 Washington Blvd., St. Louis.
General Secretary: Dr. Paul H. Holinger, 700 N. Michigan Ave., Chicago 11.
Place: Hotel Statler, Washington, D. C. Time: May 5-10, 1957.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. John R. Lindsay, Chicago.
Secretary: Dr. Hugh A. Kuhn, 112 Rimbach St., Hammond, Ind.
Place: Chicago. Time: June 11-15, 1956.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Algernon B. Reese, 73 E. 71st St., New York 21.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave., Bldg., Rochester, Minn.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

AMERICAN BOARD OF OTOLARYNGOLOGY

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Place: Sheraton-Mount Royal Hotel, Montreal, Canada. Time: May 6-11, 1956.

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SECTIONS:

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Place: The Statler Hotel, Boston. Time: Jan. 13, 1956.

Southern.—Chairman: Dr. J. M. Robison, Houston, Texas.

Place: The Shamrock, Houston, Texas. Time: Jan. 27-28, 1956.

Middle.—Chairman: Dr. William H. Craddock, Cincinnati.

Place: The Netherlands Plaza, Cincinnati. Time: Jan. 16, 1956.

Western.—Chairman: Dr. David D. De Weese, Portland, Ore.

Place: County Medical Society Building, San Francisco. Time: Jan. 21, 1956.

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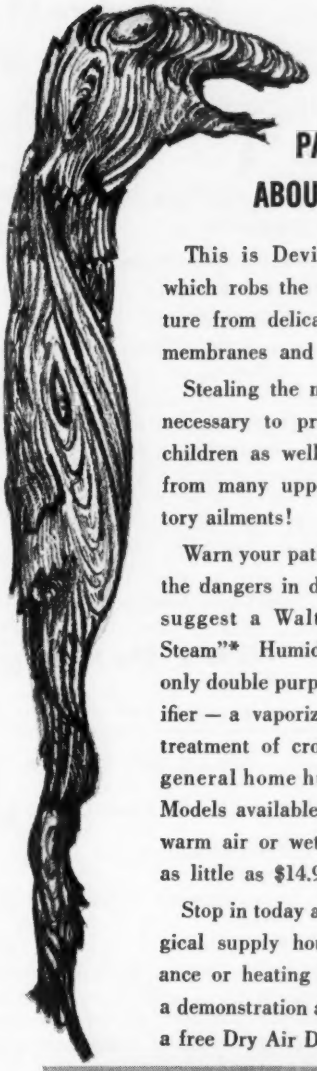

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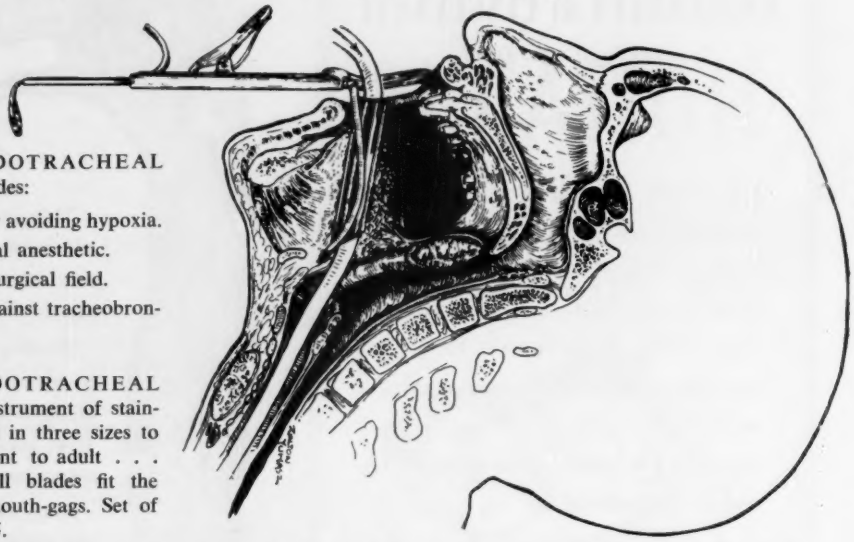

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* Barton, R. T.: Instrument for endotracheal anesthesia during oro-pharyngeal surgery. J. A. M. A. 153:1017 (Nov. 14) 1953.

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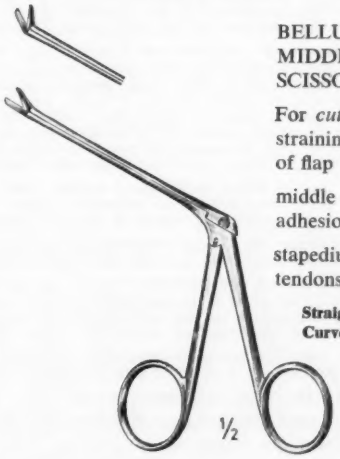

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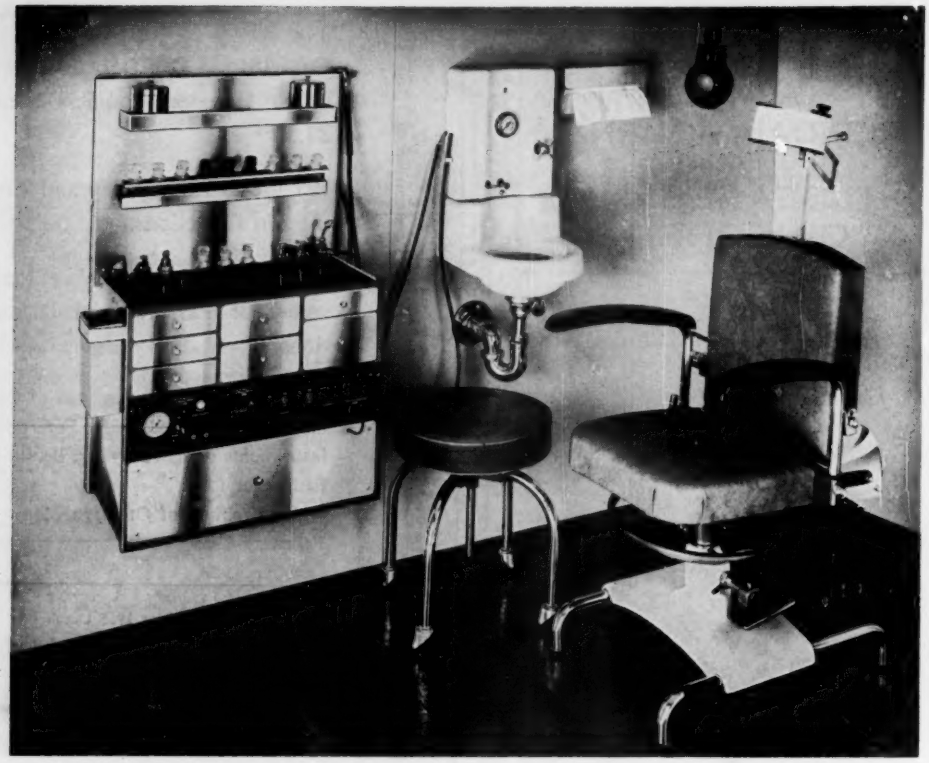

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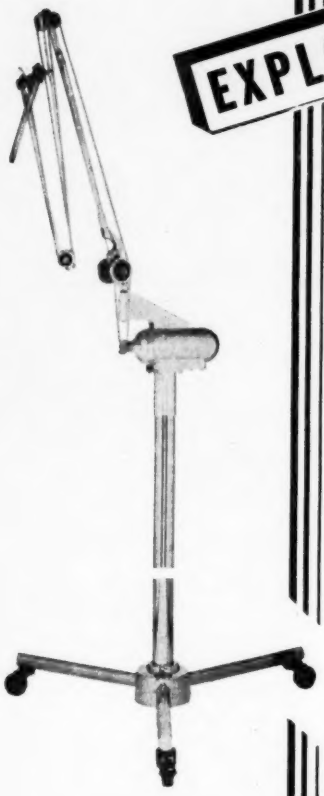

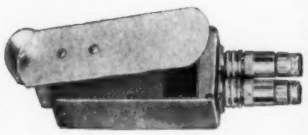

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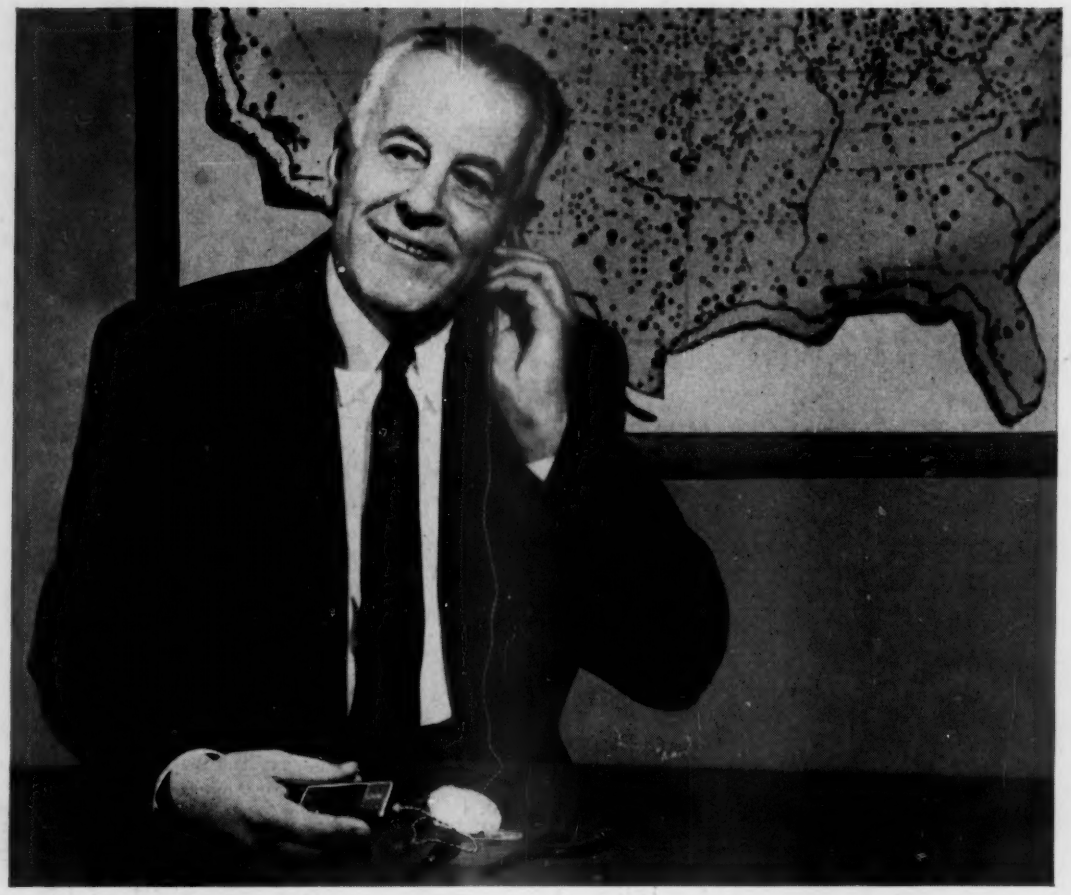

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